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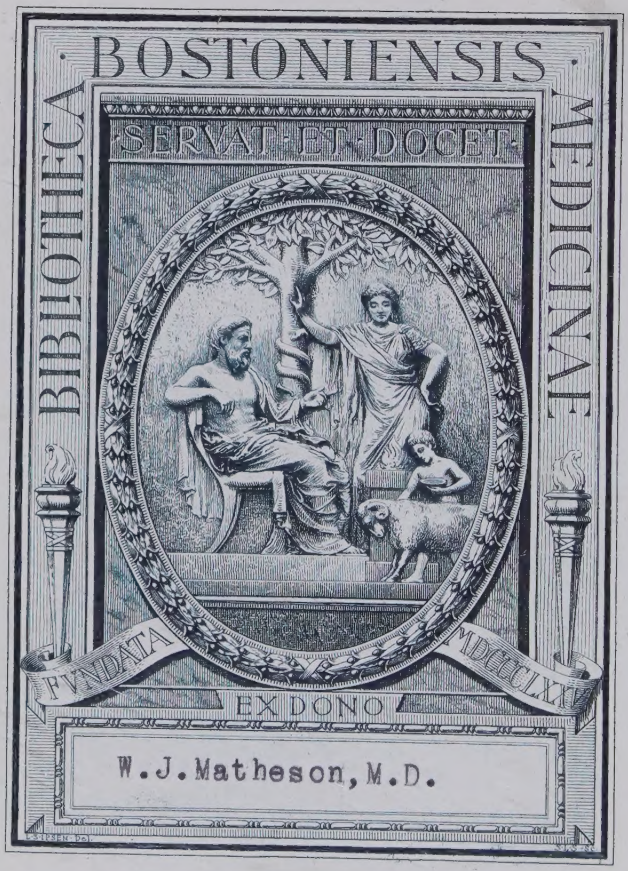
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EPIDEMIC ENCEPHALITIS

EPIDEMIC ENCEPHALITIS

ETIOLOGY · EPIDEMIOLOGY TREATMENT

REPORT OF A SURVEY BY THE
MATHESON COMMISSION

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FOREWORD

A great deal of work has been and is now being done throughout the world on the subject of encephalitis. It seemed worth while for someone to collect and tabulate this work so that the collection would be available to all who are interested. This idea so appealed to William J. Matheson that he generously undertook to make it possible. A Commission was appointed which would represent the laboratory, clinical and epidemiological viewpoints. The actual detailed work has been carried out by Dr. Josephine B. Neal, whose long experience in this and allied fields has preëminently fitted her for the position.

At Mr. Matheson's suggestion the Commission is continuing this work on encephalitis and has undertaken a number of studies of the causes, treatment and end result of this condition. Results of these studies will be published from time to time.

The Commission desires to express publicly its deep appreciation of Mr. Matheson's generous support and his very active interest and advice. His experience in chemical research and his active imagination have made his advice and constructive criticism of great value.

WILLIAM DARRACH, M.D.
Chairman

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INTRODUCTION

The word "survey" is used with such widely different meanings that it is necessary to explain briefly what we have tried to do in this Survey and the manner in which we have worked. Our object has been to collect and, so far as possible, to correlate the work done on the etiology and treatment of epidemic encephalitis and to bring together the data available on its epidemiology. To this end a bibliography has been prepared, the references being collected from the Index Medicus, the Quarterly Cumulative Index, the Catalogue of the Surgeon General's Library and the bibliographies of the more important publications.

A fairly large number of references to closely allied subjects, such as post-vaccinal encephalitis, herpes virus, and spontaneous encephalitis in rabbits, has been included.

In spite of an earnest effort to make the bibliography complete and accurate, it is certain that there are some omissions and some errors. Not all articles are listed in the different indices consulted, and articles with other titles may contain more or less data on epidemic encephalitis. The human equation makes it impossible to avoid certain minor errors in copying and transcribing references.

The references contained in the bibliography have been abstracted, with the exception of a few that were not available. The abstracts have been prepared with the intention of presenting in detail work on etiology, treatment and epidemiology. Material on the clinical aspects, pathology, etc., has been referred to briefly, so that anyone reading the abstracts would be able to learn the scope of the articles and could, therefore, consult the original papers for the material he wished to find.

In preparing the bibliography and in making the abstracts

we wish to acknowledge our indebtedness to the Bibliographical Department of the New York Academy of Medicine. The very helpful coöperation of the members of this department has been invaluable to us.

In addition to this study of the literature, a fairly wide correspondence has been established with workers on epidemic encephalitis in various parts of the world. Through this correspondence valuable data have been received, often collected with no little trouble by those to whom we have written. We wish to thank those who have so generously helped us in our work.

Personal interviews have also been held with various workers, both in this country and abroad. Practically all the subjects taken up in these interviews are included in the abstracts of articles written by those with whom we have talked. With a view to establishing contact with physicians abroad, a short visit was made to England, The Hague, Brussels and Paris in the spring of 1928.

In London various hospitals and institutions were visited: the Northern Hospital, Winchmore Hill; the Hither Green Hospital; Middlesex Hospital; National Hospital for Diseases of the Nervous System; Western Hospital; Lister Institute; the National Research Council; and various offices of the Ministry of Health.

A visit was made to Sheffield, where Dr. Arthur J. Hall devoted a great deal of time to presenting patients who had suffered from epidemic encephalitis in the Sheffield outbreaks, both patients who were attending his clinic and those who were at the hospital for mental diseases at Wadsley. The experimental work done at the Sheffield Medical School was explained by Dr. Douglas, and very beautiful pathological preparations were shown by Dr. Eaves.

Owing to the lack of fresh clinical material, no active work on the etiology of epidemic encephalitis was being carried on in England in the spring of 1928. The various physicians who had been interested in this subject were generous in giving time for discussions. To Sir Charles Martin and Dr. Ledingham of the Lister Institute, Dr. Perdrau, Dr. McIntosh, Dr. McCartney, Dr. Parsons, Dr. MacNalty, Dr. Borthwick, Dr. Greenfield, Dr. Roques, Dr. Rolleston and others, we are indebted for many courtesies and much help. Dr. Perdrau and Dr. McIntosh showed excellent pathological specimens of epidemic encephalitis and post-vaccinal encephalitis and discussed their experimental work on these diseases.

At The Hague, Dr. Bastiaanse gave an excellent demonstration of the lesions in post-vaccinal encephalitis, and Dr. Terburgh and Dr. Jitta furnished valuable data on the incidence of epidemic encephalitis in Holland.

In Brussels, Dr. Bessemans and Dr. Van Boeckel gave very generously of their time to a discussion of epidemic encephalitis and allied conditions, and also furnished valuable data. At the Pasteur Institute at Brussels, Dr. Bordet and Dr. Le Fèvre de Arric discussed the work that had been done there.

In Paris, considerable time was spent at the Pasteur Institute, where Dr. Levaditi was beginning new researches, with funds furnished by Dr. Matheson, along the lines on which he has worked for several years. Dr. Levaditi exhibited beautiful preparations of experimental encephalitis in rabbits and monkeys, and also of spontaneous encephalitis in rabbits. Patients with epidemic encephalitis treated by Dr. Levaditi's vaccine were shown at the Hôpital Cochin by Dr. Levaditi and Dr. Fournier. Interviews were also held with Dr. Roux, Dr. Martin, Dr. Calmette, Dr. Besredka, Dr. Pettit and Dr. Netter.

The Hôpital de la Salpêtrière was also visited, where there were many patients in the chronic stage of epidemic encephalitis. Dr. Guillain and Dr. Bertrand were interviewed and also Dr. de Jong of Amsterdam, who was at the hospital at the same time.

A visit was made to the Hôpital des Enfants-Malades, where Dr. Nobecourt was attending physician.

At Dr. Matheson's suggestion, a conference was held at the Medical Center in New York, at which Dr. E. C. Rosenow presented his work. At the conference were the members of the Commission and also Dr. Jobling, Dr. Homer Swift, Dr. Dochez, Dr. Peter Olitsky and Dr. Tallman.

Dr. Rosenow read in part from his paper: "Localization in Animals of Streptococci from Cases of Epidemic Hiccup, Encephalitis, Spasmodic Torticollis and Chorea," published in the *Archives of Neurology and Psychiatry,* 19: 424, 1928, and also presented some new and unpublished work on poliomyelitis. His lecture was illustrated by lantern slides and moving pictures and at the close he exhibited rabbits inoculated the previous day with naso-pharyngeal secretions of cases of encephalitis and poliomyelitis. He also showed under the microscope preparations of cultures and smears of spinal fluids.

Following the lecture and demonstrations, there was a discussion by the members of the Commission and the physicians who had accepted Dr. Darrach's invitation to be present.

In order to investigate further the comparative merits of the filtrable viruses and the cultivable bacteria as the etiologic agents of epidemic encephalitis, Dr. Matheson persuaded Dr. Levaditi to visit Dr. Rosenow's laboratory to study his work and methods. Dr. Levaditi will carry on experiments, as a

result of this visit, the reports of which will not be available for some time.

Dr. Charles R. Stockard, who, in connection with his research work, has a kennel for dogs under expert observation and conducted along highly scientific lines, has noted in dogs suffering from different intestinal parasites certain manifestations of disturbances of the central nervous system. Assisted by a grant from Dr. Matheson, Dr. Stockard is making a careful investigation of the cause and nature of these neurological manifestations to learn whether or not they may have a bearing on any form of encephalitis in man.

The bibliography has been prepared by Jean N. Baker, the chapter on epidemiology by Dr. Helen Harrington, and the chapters on post-vaccinal encephalitis, etiology and treatment by Dr. Josephine B. Neal. In preparing the chapters on epidemiology, post-vaccinal encephalitis and etiology, it has been our aim to present an unbiased summary of the contributions of the different authors. No attempt has been made to criticize the work or to draw conclusions designed to express our own opinions.

All the members of the Commission have given most generously of their time in connection with the making of the Survey and the preparation of the monograph. Dr. Emerson has made many valuable suggestions in regard to the scope and the details of the work on epidemiology. The chapters on post-vaccinal encephalitis and etiology were submitted to Dr. Park and to Dr. Gay, whose knowledge of these subjects enabled them to give helpful suggestions and advice. The chapter on treatment was thoroughly discussed with Dr. Tilney. The wide experience that Dr. Tilney has had in treating patients with epidemic encephalitis has made his estimations of the various therapeutic measures of great value.

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Chapter I

SUMMARY OF INVESTIGATIONS ON THE ETIOLOGY OF EPIDEMIC ENCEPHALITIS

Early Theories of Epidemic Encephalitis

The difficulties in diagnosing epidemic encephalitis, especially great before its establishment as a definite clinical entity, quite naturally led to its confusion at first with other diseases. Much valuable work in clarifying this situation was done by the Ministry of Health in London (5). The earliest cases of the disease described in England were observed by Dr. Arthur J. Hall (H 22) and Dr. Wilfred Harris (H 74). Dr. Harris called attention to certain points of similarity in his cases to the clinical picture of "botulism." In consequence of this, the Medical Research Committee undertook at once a complete bacteriological investigation, in coöperation with medical practitioners and medical officers of health. The study included not only a consideration of "botulism"; it also covered the question of infection or poisoning from food stuffs—including the absence from the food of some essential element—or toxic agents, such as benzene used in the manufacture of oleomargarine, and solanin (an alkaloidal glucoside) in undue concentration in potatoes; also researches upon lathyrism, as well as upon pellagra, beriberi and other "deficiency diseases."

The results of the inquiry are summarized as follows:

"First, on the bacteriological side, Dr. McIntosh, of the London Hospital, who was appointed to carry out the investigation on behalf of the Medical Research Committee, failed entirely to recover the *Bacillus botulinus* from tissues and fluids of fatal cases and from articles of food which had been regarded with suspicion; he failed also to obtain

evidence that serum from recovered patients agglutinated this bacillus or protected mice from the effects of its toxin; and he found microscopical evidence of changes in the nervous system which pointed to an acute infective disease, rather than to an intoxication. Secondly, on the epidemiological side the enquiries by Drs. Hancock and Pearse showed that among the 58 cases investigated up to May 7th there was no instance of more than one case in a household, that many of the patients had not eaten any food which might reasonably be regarded with suspicion; that in instances in which patients had eaten such foods it was almost invariably without ill effect; and finally, that in at least two cases the patients attacked were infants exclusively breast-fed."

This investigation seems to disprove quite definitely the possibility of epidemic encephalitis being caused by infection or poisoning from foods. But in spite of this, Veillard (V 41a) in 1923, Bouquet (B 318) and Séval (S 157-S 159) in 1927 and 1928 advanced the theory that encephalitis is caused by poisoning with small amounts of hydrocyanic acid in flour containing French beans, or by poisoning with the chick-pea (lathyrism), or that it is a condition due to the use of polished rice (beriberi) or corn (pellagra). Séval goes on to say that an intercurrent febrile infection releases the toxin of the ingested food stuffs.

The spread of the disease to so many nations with such varying dietary habits speaks strongly against such a theory.

Another early theory advanced was that these cases might be more or less unusual forms of acute poliomyelitis. An investigation of this possibility was therefore undertaken by the Ministry of Health (5). Professor G. Marinesco made a histological study of specimens from fatal cases, and Dr. McIntosh carried out both histological studies and inoculation

of animals. Their researches supported the clinical and epidemiological evidence that the two diseases were not identical. To quote from the report: "As a result of their separate researches both Professor Marinesco and Dr. McIntosh arrived independently at the conclusion that encephalitis lethargica, as it appeared in the present outbreak, is identical with the illness described by von Economo in Austria and Professor Netter in France, and that it is a disease sui generis, anatomically and clinically distinct from analagous affections."

Certain points of difference between poliomyelitis and epidemic encephalitis are brought out by Tilney and Howe (T 60), who conclude the comparison with the following paragraph:

"Possibly the greatest difference between the two conditions is in the nature of the action of the virus on the nerve elements. In poliomyelitis there is a sudden, sharp attack which develops rapidly and in a few days attains the maximum of severity, quickly producing the greatest extent of destruction. In epidemic encephalitis the onset is frequently very gradual and the progress of the disease slow, in some instances requiring several months to reach its greatest severity, and even then the findings have a tendency to variability, being subject to exacerbations and remissions."

A most important point in proving that epidemic encephalitis is not a form of poliomyelitis was made by Amoss (A 100), who showed that serum from convalescent cases of epidemic encephalitis had no immunizing power for poliomyelitis virus.

A brief summary of the points of difference of these two diseases follows: Poliomyelitis is an acute disease, attacking young children chiefly. Its greatest prevalence is usually in hot weather. It can be transmitted to monkeys with a fair

degree of ease. Epidemic encephalitis is essentially a chronic disease, attacking people of all ages. Its greatest prevalence is usually in the winter months. Most attempts to transmit it to animals have resulted in failure. The two diseases differ histologically, especially in the localization of the lesions. Serum from convalescent cases of epidemic encephalitis has no immunizing power for poliomyelitis virus.

At the present time, certainly, the great majority of physicians believe that these diseases are separate clinical entities. But it is quite possible that in the future the classification of poliomyelitis may need to be revised and that certain rare cases, previously diagnosed as the encephalitic type of poliomyelitis, may be considered cases of epidemic encephalitis.

While this opinion is held by most physicians, there is a group who hold a different theory. One of the ablest exponents of the latter is Crookshank (C 282), who wrote: "The synthetic view, then, is that encephalitis lethargica represents a nervous form of influenza that tends to occur in older, and epidemic poliomyelitis a like form that is more frequent in younger patients." This certainly connotes in his opinion a close relationship, not only between poliomyelitis and epidemic encephalitis, but also between epidemic encephalitis and influenza.

The question of the possible relationship of epidemic encephalitis and influenza has been widely discussed, and diametrically opposite opinions are held by qualified investigators. For example, monographs on influenza have been prepared by Crookshank and by Jordan.

Crookshank (C 282a), reinforcing his opinion with a wealth of historical references, concluded:

"(1) Clinical occurrences of the nature that we now ascribe to forms of encephalo-myelitis, or meningo-encephalomyelitis,

have been recorded in modern times for at least 450 years. (2) In great part these occurrences have been noted as incidental to major prevalences, known historically as the sweating sicknesses, the epidemic catarrhs, influenzas and the like. (3) Special prevalences of these occurrences have also been described as manifestations of special diseases. These special prevalences have usually appeared shortly before or shortly after major 'influenzal' epidemics, or else in geographical proximity to endemic-epidemic and endemic influenzal prevalences. (4) Epidemic meningo-encephalomyelitis represents an intensive and specialized reaction that has the same epidemiological relation to pandemic influenza as have the prevalences and epidemics of 'septic' pneumonia, of gastro-intestinal illness, and of other maladies described as occurring before and after the wide diffusions generally referred to as pandemic influenza. (5) Owing to the relative infrequency of epidemic encephalomyelitis and its marked variation in type, historical investigation is necessary in order that contemporary occurrences be viewed in a correct perspective."

On the other hand, Jordan (J 58a), after a careful epidemiological study of the two diseases, was not impressed either with their time relationship in different outbreaks during the past ten or twelve years, or with the possibility of identifying the disease we now call epidemic encephalitis with the clinical description of eidemics of disease of the nervous system occurring many years ago. He summarized his opinions as follows:

"1. It does not seem possible to identify surely the present pandemic of encephalitis with nervous disorders accompanying or following the historical pandemics of influenza. In point of fact, no unusual prevalence of encephalitis appears to have been noted in the United States or in northern Europe during

the influenza pandemic of 1889-1890, and the form of encephalitis that was occasionally observed seems to have been different clinically and anatomically from the present epidemic disease. 2. The epidemics of encephalitis in the United States from 1918-1923 showed in some places a certain correspondence with the recurrent waves of influenza, but it is at least doubtful whether this means anything more than that both diseases were introduced about the same time and that both thrive commonly though not exclusively in cold weather. As set forth above, a detailed comparison of the reported cases of the two diseases in New York and Chicago does not strengthen the hypothesis of a genuine relationship. The same lack of correspondence has occurred in Great Britain, Portugal and other European localities. 3. Until a coincidence or close sequence of the two diseases has been more convincingly demonstrated, it seems premature to discuss the existence of a 'neurotropic' influenza virus or to speculate on the 'activation,' of an encephalitic virus by an influenza virus, or on the nature of the encephalitic component of a 'complex grippe virus.' 4. A coincidence in prevalence does not prove a common cause. Even should a close time correspondence be established, it would not follow that the relation was other than an increased susceptibility to one infection produced by another unrelated infection."

Crookshank (C 282) referred to the work of Tarozzi and Rosenow in confirming his conception of the relationship of influenza and encephalitis. Rosenow's work, which will be described in detail later, would indicate a close relationship in influenza, poliomyelitis and epidemic encephalitis, since in all three diseases he claims that green-producing streptococci, with very similar serological reactions, are the causative agents. It may be stated that Rosenow (R 254) emphasizes the finding

of the streptococcus in the lesions in the brains of fatal cases, thereby implying, at least, that the actual invasion of the brain is the cause of the disease, though in a later article (R 257) he stated that the specific localizing power of the streptococcus is in part dependent on a toxin or poison. Tarozzi (T 12), on the other hand, attributed the histological changes in the central nervous system to the local action of a circulating toxin produced by diplostreptococci, which cause an inflammation in the respiratory tract. He further claimed that histological changes similar to those in epidemic encephalitis may be produced experimentally in rabbits by the subdural, intraperitoneal or intravenous injection of live culture, killed culture, or toxin of diplostreptococci from acute broncho-pneumonia of the influenzal type.

Hall (H 29a) pointed out that whereas influenza is highly contagious and has, in time of epidemics, a high morbidity rate, epidemic encephalitis is exactly the reverse in these two details. He also commented on their lack of resemblance clinically and in the number and severity of their residua.

The question of their identity or close relationship is further complicated by the fact that influenza undoubtedly has a toxic effect on various parts of the central nervous system. Jelliffe described these effects in detail in a chapter in Crookshank's "Influenza." But he said: "Cerebral involvements in influenza have been noted from the earliest times. Mild involvements are frequent, severe ones comparatively rare." However, it is rather generally conceded that influenza, like measles and other acute infections, may be followed occasionally by encephalitis. While Tarozzi and others claim that there is no histological difference between epidemic encephalitis and influenzal encephalitis, Strümpell (S 389), whose experience includes the epidemic of influenza in 1889-92 and

also the recent outbreak of influenza and epidemic encephalitis, considers that epidemic encephalitis is distinct from influenzal encephalitis. He stated that the characteristic histological picture of influenzal encephalitis is a circumscribed punctiform hemorrhagic encephalitis localized chiefly in one hemisphere, whereas in epidemic encephalitis the gross anatomical changes are minor, consisting in hyperemia and slight swelling. Gross (G 152) also stated that epidemic encephalitis is anatomically distinct from the cases of influenza encephalitis and grippe encephalitis described by Strümpell and Leichtenstein in the epidemic of 1889-90.

MacNalty (M 23) stated that "no reliable evidence in favour of the identity of influenza and epidemic encephalitis is forthcoming," and he concluded that it is a distinct and separate disease.

Flexner (F 71) is strongly of the opinion that epidemic encephalitis is an entirely distinct clinical entity with no direct relationship to influenza. He wrote: "Let it be recalled that the first cases of the disease (epidemic encephalitis) were observed in Europe in 1916 and 1917. Now, the epidemic of influenza which reached a peak in 1918-19 was already on the march in Europe as early as 1917, and probably earlier. An overlapping in time is therefore apparent. Could epidemic encephalitis be merely one manifestation, albeit a severe one, of epidemic influenza? That it is such a manifestation merely has been repeatedly asserted. The relationship has been as assiduously denied as upheld. What, then, are the evidences for epidemic encephalitis being a disease distinct from influenza, or, as we say, a disease by itself?

"An argument could be based on the circumstance that while the two diseases overlapped in time, as in 1918-19, once the pandemic of influenza had passed, that of epidemic

encephalitis still continued. The height of the epidemic wave of epidemic encephalitis came several years after that of the influenza wave, as in England, for example, in 1924-25. The moment has not yet arrived at which to determine finally the etiologic relationship, if any, existing between influenza and encephalitis, and can scarcely come until our knowledge of their immediate sources or microbic incitants becomes definite.

"Although we are denied the answer we seek by direct inquiry, we are offered it through a somewhat devious path. The pandemic of influenza of 1889-92, the one immediately preceding that of 1918-22, was to all intents and purposes as well studied clinically, pathologically, and bacteriologically as could be done now. In view of the state of development of scientific medicine in 1890, it is not to be doubted that, if large numbers of clinical cases corresponding to those we now call epidemic encephalitis had arisen, they would have been recognized and described. Their occurrence is indeed conspicuous through absence, although other usually less severe and impressive instances of nervous complications of epidemic influenza are frequently described as constituting part of the influenzal disease of the nineties. We are therefore left in the position of dealing with epidemic encephalitis as a disease having its own particular origin or etiology."

It is apparent that the weight of opinion is against either the identity of influenza and epidemic encephalitis, or a direct relationship between the two diseases.

Many authorities, however, consider favorably the possibility of some indirect relationship. Hall (H 29a) expressed himself as of this opinion. Flexner (F 64) wrote: "Whether an attack of the first acts to predispose to an attack of the second in the manner of the predisposing effect of influenza on

bacterial affections of the respiratory tract, is another question awaiting close study." Kramer (K 114) is of the opinion that influenza is one of the factors, many of which are unknown, that are responsible for the epidemic spreading of the sporadic cases of encephalitis. Zinsser (Z 14a) wrote: "Nevertheless, it must be borne in mind, especially in connection with the facts related below, that a great deal of evidence supports the view that the coincidence of influenza and encephalitis may be causally related, one disease rendering in some manner susceptible to the other."

Since the outbreak of epidemic encephalitis there has been more or less comment, especially by non-professional people, on the possible occurrence of a similar disease in animals. It may be of interest, therefore, to refer briefly to this subject.

During the years that epidemic encephalitis has been prevalent, accounts have been published of diseases in animals that have suggested more or less the clinical picture of epidemic encephalitis in man.

Reports of such outbreaks, affecting dogs and cats in particular and also horses and ferrets, were described by Parsons in 1922 (P 47a). Most of these were investigated and little evidence was collected to show that these diseases in animals had any connection with epidemic encephalitis, except a coincidence of time.

Donatien and Bosselut in 1922 (D 145) published an account of the outbreak of an acute infectious disease in cows in the region of Algiers. Calves, sheep, rabbits and guinea pigs were inoculated with emulsion of the spleen and bulb. All the animals except the sheep were affected. The disease was transmissible in series in the cow, rabbit and guinea pig. No organisms were demonstrable by culture.

Pugh in 1926 (P 222) reported an outbreak of a disease

known as the nervous form of distemper in dogs. It was stated that both the clinical picture and the microscopic lesions in the brain strikingly resembled epidemic encephalitis. No experimental work was done. It was pointed out that distemper is now considered due to a filter-passing virus.

Wright in 1927 (W 148) described the symptoms, lesions and bacteriology of canine meningo-encephalitis, sometimes called Fright's disease. A short Gram-negative bacillus, growing well on ordinary media, was isolated from the spinal fluid, spleen and liver of these dogs. This organism was pathogenic for dogs and guinea pigs, and was considered by the author to be the probable cause of the disease. Other workers have assumed that errors of diet or intestinal parasites were the cause. No connection with epidemic encephalitis was claimed by the author.

A list of diseases considered due to filtrable viruses, published by Rivers (R 134a), shows a rather large proportion occurring in animals. Several of these affect the central nervous system. It is not surprising, therefore, that there occur in animals symptoms and pathologic lesions more or less resembling epidemic encephalitis in man, the latter being believed by many to be caused by a filtrable virus.

MacNalty in 1927 (M 23) gave a very complete account of epidemic nervous diseases in animals.

A topic which belongs rather to the realm of pathology than to that of etiology, but which seems to deserve a brief consideration here, is that of the various "minute bodies," or cellular inclusions.

These were first described by Da Fano and Ingleby (D 11) in 1918-19, in the nerve cells of three cases. Later (1921) Da Fano (D 5) found them in the salivary glands, as well as in the nerve cells, and suggested that they might

represent a living agent, the cause of encephalitis, or that they might be products of degeneration, or even optical illusions. In 1923 (D 7) he described similar forms in rabbits with experimental herpetic meningo-encephalitis. He considered them very similar to the "neurocorpuscles encephalitiques" described by Levaditi, Harvier and Nicolau (L 197). He went on to say that the smallest of these granular structures may be the virus itself, or particles of organic matter to which the virus clings. He also described similar bodies in the brain tissues of fatal cases in 1924 (D 8), working both alone and with Ingleby. A rather complete discussion of this subject is given by Levaditi (L 174) in 1922.

Somewhat similar bodies were described by Volpino and Desderi (V 103); Volpino and Graziadei (V 104), who also described forms similar to, but not identical with, amylaceous bodies; Ottolenghi, d'Antona and Tonietti (O 46), in animals inoculated with their virus; Urechia (U 8); Shaw (S 164); Mittasch (M 320); Meleney (M 250), Guiraud in 1922 (G 198) and 1923 (G 199); Lucksch (L 333), who stated that they are without significance, as they are found in brains of persons dying of other diseases; Joest (J 48), who pointed out their resemblance to inclusion bodies found in the ganglion cells in rabies, chicken-pest, distemper of dogs, Borna's disease in horses, and sheep encephalitis—all diseases due to a filtrable ultra virus; Omorokow (O 31); Modena (M 329); Goodpasture (G 101), who discussed the nuclear changes in the ganglion cells in experimental herpetic encephalitis; Dechaume (D 54), who described the cell inclusions in the brain which he, and other workers, compared to Negri bodies, and, in his second article (D 56), mentioned somewhat different cell inclusions in the liver and kidney; and Alberca-Lorente (A 74), in experimental encephalitis. Parker (P 40),

on the contrary, denied the existence of these intranuclear bodies in cases of human epidemic encephalitis and regarded it as an argument against the relationship between encephalitis and herpes.

Rivers (R 134a) commented upon this subject as follows: "The 'minute bodies' discovered by Da Fano and Ingleby have proved rather puzzling. They were found in the cytoplasm of cells like lymphocytes and small neuroglial cells and also extracellularly. They stain intensely with basic dyes and in the illustrations seem to be surrounded by halos. In herpetic encephalitis, Da Fano has observed bodies which he terms 'herpetic minute bodies.' He concludes that these are the virus itself 'or particles of organic matter to which the virus tenaciously clings.' Nicholson and I have been able in our own experiments (15) to identify the herpetic minute bodies and there is no question that in Giemsa preparations they have a suggestive organismal appearance. On bleaching the preparations, however, it was found that they are merely small particles of brownish pigment which resist the solvent action of strong hydrochloric acid. It is probable that the minute bodies in epidemic encephalitis are of the same nature but this cannot be asserted definitely until their solubility has been tested."

When we consider the many years that the nature of the Negri bodies (to which the inclusion bodies of epidemic encephalitis seem to bear some resemblance) have been discussed, without the question being solved, it is not surprising that the significance of these inclusion bodies remains an open question.

The three most important theories in regard to the etiology of epidemic encephalitis are: (1) That it is a toxic disturbance of the central nervous system; (2) that it is caused by cultivable bacteria; and (3) that it is caused by a filtrable virus.

The principal investigations along these three lines will be presented.

Toxic Disturbances of the Central Nervous System

The theory that epidemic encephalitis is a toxic disturbance of the central nervous system subdivides itself into two possibilities: (a) That the toxins are due to bacteria located possibly in the gastro-intestinal or the respiratory tract; and (b) that the toxins are elaborated as the result of metabolic disturbances. This theory is developed partly from positive, partly from negative, lines of reasoning. It is well known that very similar clinical and pathologic pictures may be produced by the action on the brain of certain toxins of bacterial origin, such as those of typhus and *B. botulinus,* and by chemical poisons, such as lead. A toxic origin of the disease would fit in very well with the large percentage of negative results obtained in attempts to discover the etiologic agent by cultural methods or by animal experimentation.

Against this theory is the manner in which the disease appeared rather suddenly, spread in increasing numbers from place to place, often with recurring waves, and has since subsided, this strongly suggesting that it is of an epidemic nature. In its epidemiologic manifestations it bears a strong resemblence to poliomyelitis and to meningococcic meningitis. Therefore, if one assumes that it is of a toxic nature, one is forced to hypothesize some infectious disease that was prevalent in different parts of the world for some time and has since subsided. It is, moreover, a little difficult to conceive of a focal infection in the respiratory or gastro-intestinal tract persisting for so many years and elaborating toxins that so profoundly affect the central nervous system without giving more generally recognized local signs in the infected areas.

Martin (M 145), in 1918 was perhaps the first to suggest that a toxin elaborated by organisms in the nasopharynx might slowly localize in the central nervous system, producing the lesions observed, and that, therefore, the etiologic agents should be looked for in the nasopharynx. He called attention to Sainton's (S 22) observations that there frequently occurred in the pharynx of patients suffering from epidemic encephalitis, ulcerative lesions similar to those found in grippe.

Tarozzi (T 12), who has already been cited as believing that epidemic encephalitis cannot be distinguished from the encephalitis that may accompany influenza, stated that in epidemic encephalitis he found the same alterations in the respiratory tract that are found in broncho-pneumonia of influenzal type, and expressed the opinion that the histological alterations of the central nervous system are presumably to be attributed to the local action of circulatory toxins produced by diplo-streptococci, which cause inflammation of the respiratory tract. He further stated that histological changes similar to those observed in epidemic encephalitis can be produced experimentally in rabbits by injecting subdurally (and in some cases intraperitoneally or into the jugular vein or carotid artery) live cultures, killed cultures, or toxins of a diplo-streptococcus from acute broncho-pneumonia types of influenza.

Yates and Barnes (Y 4), although apparently believing that the cause of epidemic encephalitis is a filter-passing organism, advanced a hypothesis that would also fit in with the theory of a toxin. They found in most of their cases of epidemic encephalitis evidence of sinus involvment which they thought increased the permeability of the mucous membrane to toxins, or to the virus of epidemic encephalitis, and was an important factor in the development of the disease.

Beck (B 97) stated that a large number of diseases of the nervous system are due to the selective action of toxins on neurones. Rosenow has shown the neurotropic action of a steptococcus in his experimental study of epidemic encephalitis. In patients with residual encephalitis there are usually glaring foci of infection. It seems reasonable to believe that the original neurotropic organism has found refuge in these foci.

Salzman (S 40-S 41), in two articles, advanced the opinion that epidemic encephalitis is not an infectious and epidemic disease, but rather that it is of toxic origin, the toxins, with a selective action on the central nervous system, being elaborated in some focus of infection in the body, among these the gall bladder. He referred to six cases with gall bladder involvment, which were operated on with subsequent recovery. All cultures from the gall bladder were sterile.

These are the principal references to toxins of bacterial origin as the etiologic factor in epidemic encephaltis. It is, however, not possible to separate clearly the theories of bacterial toxins and toxins arising from metabolic disturbances, since in the production and effects of metabolic disturbances bacterial infections may play a more or less prominent part.

Buscaino (B 441) advanced the theory that the post-encephalitic amyostatic syndrome is a degenerative lesion of the central nervous system due to the presence in the circulation of special toxic substances, particularly aromatic amines due to hepatic lesions, which, he stated, are the rule in the evolution of this disease. He was uncertain as to whether or not it is a question of specific bacterial action. In a later article (B 442) he reiterated this opinion and added that lesions in the intestinal barrier also may play a part in the passage of these toxins into the circulation. In 1928 (B 443) he em-

phasized the rôle of intestinal lesions in the development of the chronic stage of the disease, describing the necropsies of two patients with grave lesions of the intestines and pancreas. In this contribution he states that the most probable hypothesis is that at the time of the encephalitic infection, with localization in the mesencephalon and basal ganglia, lesions of the gastro-intestinal mucosa develop, varying according to the susceptibility of the patient. If these are not cured, they engender the post-encephalitic syndrome.

Lewy and Tiefenbach (L 250) produced encephalitis in rabbits by mixing manganese dioxide with their food.

Fuchs (F 156) described the production of encephalitis in cats and dogs by the injection of guanidin. He referred to earlier experiments in which he showed that, under the influence of pathogenic micro-organisms in the body, guanidin might be produced in pathological quantities.

The same clinical and histological picture was produced by feeding meat to dogs after the production of Eck's fistula. He stated that these experiments open the way to the understanding of Wilson's symptom complex, a chronic degenerative process in the liver parenchyma and degenerative processes in the lenticular nucleus.

Pollak (P 181) studied the histological picture in Fuchs' experiments.

Silberstein, working with Hoff and Orel (S 216, H 191, H 192, O 37, S 217), corroborated Fuchs' experiments in developing encephalitis in dogs after the production of Eck's fistula. They concluded, however, that the encephalitis was caused by a virus, probably rabies, in the nasopharynx of the dog, which was harmless until the metabolic disturbance injured the protective mechanism of the central nervous system.

In articles by Stern and Meyer-Bisch in 1922 (S 338) and

by Meyer-Bisch and Stern in 1923 (M 291) it is stated that urobilinuria was found in cases of chronic, progressive epidemic encephalitis, and also in certain cases in the acute stage. They also found a marked disturbance of carbohydrate metabolism. They do not believe that the lesions in the liver are the result of the lesions in the central nervous system, but rather a possible factor in the development of these lesions of the central nervous system. They cite for purposes of comparison Wilson's disease, the cerebral changes in acute yellow atrophy of the liver, and Fuchs' experiments with Eck's fistula in dogs. They also state that poisons which affect the brain through the blood stream, such as carbon monoxide, manganese, and hydrocyanic acid, elect the lenticular nucleus, while local infectious diseases do not show this elective habit.

Gottschalk and von Hoesslin (G 113) carried out liver function tests in seven patients with chronic epidemic encephalitis. They found that there was no recognizable disturbance of carbohydrate metabolism. The urobilin reaction was weakly positive in two instances.

Mercio (M 264) obtained negative results in certain liver function tests in chronic cases of epidemic encephalitis, and concluded that there was no evidence of correlation between the lesions of the liver and those of the corpus striatum.

Ceconi (C 76) suggested that probably the factors of the production of the amyostatic syndrome, whether they are infectious, toxic, or a combination of the two, are not dependent for their origin on the liver nor on the nervous system, but that they act at the same time on both the liver and the central nervous system.

Runge and Hagemann (R 301) found a pathological liver function in twenty cases of epidemic encephalitis. They concluded, in agreement with Meyer-Bisch and Stern, that the

significance of these disturbances of liver function and their relation to cerebral disease is not determined. They raised the question as to whether the injury to the liver makes possible the penetration of toxins into the brain with an elective action on the stem ganglia.

Pedrinoni (P 66) cited Ottonello (O 50) (this reference was not available for our use), who reported no change in the liver aside from a slight increase or decrease in the area. The cases studied by Pedrinoni showed a disturbance in liver function in the majority of instances.

Bolsi (B 273) reported a case of fatty degeneration of the liver and expressed the opinion that the disturbances of the liver function and those of the central nervous system are produced by the same toxic agent.

O'Flynn and Critchley (O 18) were unable to find any clinical, histological or biological evidence to suggest a hepatic disturbance after epidemic encephalitis.

Good (G 97) advanced the opinion that the prolonged mental strain of the disease so weakens the resistive power of the nerve cells that, on the invasion of the blood stream by toxins, these cells are most affected.

Graziani (G 125) found no case of marked insufficiency of the liver and only three cases of mild involvement. He stated that the hepatic changes cannot be regarded as causal factors of the encephalitis, but are rather results of it.

Audo-Gianotti (A 156) concluded that in every case of the Parkinsonian syndrome of epidemic encephalitis there is a certain degree of insufficiency of the liver which increases with the progress of the disease. This functional liver insufficiency probably finds its cause in a "rigidity" of the hepatic cells, following lesions of the vegetative nervous system. This insufficiency of the liver, therefore, has no importance in the

etiology of the disease itself, and must be clearly differentiated from the condition found in Wilson's disease.

Seletzky (S 146) believed that Wilson's disease is chronic disseminated encephalitis (following acute epidemic encephalitis).

Considerable work has been done in studying the disturbances in metabolism and in the vegetative nervous system of patients suffering from epidemic encephalitis, especially in the chronic stage, from which the authors have not tried to draw conclusions as to whether these disturbances were the result, or the possible cause, of the disease, or as to their bearings on the question of its toxic origin. It seems worth while, however, to refer briefly to the most important of these studies. Their bearing on the question of etiology is indirect, but they are of sufficient general interest to warrant a short discussion.

Hess and Goldstein (H 164) found in chronic encephalitis a reduction in the white blood cells in the peripheral circulation, after intravenous injection of quinine, due, they believed, to a deficiency in the liver veins rather than to an alteration of the liver cells. They also studied the results of the administration of adrenalin, atropin and pilocarpin, as revealed in disturbances of innervation in Parkinsonism. They found moderate, but constant, reduction of blood cholesterol.

Kasanin and Grabfield (K 17) concluded that there is a fundamental disturbance of the sugar metabolism in this disease, and that the high spinal fluid sugar often described is probably associated with the high blood sugar.

Tkatschew and Axenow (T 72) reported a disturbance of sugar metabolism in chronic epidemic encephalitis. They attributed the disturbance of the power of assimilation of the

liver to an affection of the central regulating mechanism, probably of the mid-brain.

McCowan, Harris and Mann (M 211) stated that an increased blood and spinal fluid sugar may occur in chronic epidemic encephalitis, but that it is not necessarily characteristic of it, nor does it have disgnostic value. However, there is a definite derangement of carbohydrate metabolism.

The same authors (M 210) commented on the difference in the effects of hyoscine on normal persons, and on patients in the chronic stage of epidemic encephalitis. In normal persons there was a dulling of the senses of coördination, and a depression of the glycogenic function. In patients with Parkinsonism there was improvement clinically and in the blood sugar curve. Other chronic cases of epidemic encephalitis did not show this improvement.

Wollheim (W 133) studied and compared the blood sugar in both hyperkinetic and akinetic types of epidemic encephalitis. He believed that the origin of the change was in the central function of the vegetative system.

Raphael, Searle and Ferguson (R 16) compared the carbohydrate tolerance of cases of chronic epidemic encephalitis with the amyostatic syndrome, with other patients presenting non-infectious striate disorders. They found a certain resemblance between these two types of cases, as well as to schizophrenia.

Naccarati (N 3) studied the basal metabolism in forty-seven patients in the chronic stage of epidemic encephalitis. In twenty-eight cases (60 percent) there was a departure from the normal, twenty-five showing an increase in the metabolic rate, and three a decrease. He attributed this alteration to lesions in the vegetative centers located in the gray substance around the third ventricle and the aqueduct of Sylvius.

Shoenemann (S 110) made functional tests of internal organs in chronic cases of epidemic encephalitis. He found no definite indication of disturbed liver function, and a normal kidney function, but numerous disturbances of the vegetative nervous system.

An extensive study of the biochemistry of chronic epidemic encephalitis was published in six parts in the *Zeitschrift für die Gesamte Neurologie und Psychiatrie* in 1926. The catalase-index was studied by Brailovsky, Tschalissov and Bèrlin (B 357); the blood calcium by Tschalissov (T 108); the tonus of the sympathetic nervous system by Solowjewa and Stschedrakov (S 255); the comparison of the sympathetic system in chronic epidemic encephalitis and in schizophrenia by Maslov (M 153); the blood chlorides by Asarch (A 138); and serum lipase by Brailovsky (B 356.)

Hirst and Imrie (H 181) found a high uric acid output in severe cases of Parkinsonian rigidity. There was clinical improvement and a decrease in the uric acid following the administration of tincture of belladonna and also of hyoscine.

Holzer and Klein (H 219a) reported a case of chronic encephalitis with Parkinsonism showing various metabolic disturbances. They stated that the essential feature in the disturbance of the vegetative function seems to consist in a general torpidity of the sympathetic processes, and suggested the possibility that this is an endocrine disorder resembling myxedema.

Ohmori (O 20) compared the liver function tests in Type A encephalitis (called in Japan winter-encephalitis and believed to be identical with epidemic encephalitis), and Type B encephalitis (called in Japan summer-encephalitis and believed by some to be a different disease). In Type A, disturbances of liver functions are frequently observed, while

in Type B they are usually absent or slight. The absence of symptoms of Parkinsonism in Type B is also noteworthy. The author does not definitely conclude, however, that a genetic separation of the two types is justified.

It may be noted, in passing, that not all workers in the western hemisphere found evidence of disturbances of the liver function in the patients whom they studied.

Schargorodsky and Scheimann published in 1927 and 1928 three articles (S 84a, S 84b, S 85) on hepatic functioning and catabolism in chronic epidemic encephalitis. They concluded that patients with residual symptoms after epidemic encephalitis show genuinely disturbed carbohydrate metabolism. Hyperglycemia prevailed among the groups affected by hypertonia, and hypoglycemia among the groups affected by hyperkinesia. Prolonged hyper- and hypo-glycemia indicate functional disturbances of the system which regulate the level of blood sugar. These systems include the cells of the liver and other tissues, as well as the central nervous system.

The abnormally high nitrogen values should be attributed to increased albumin decomposition processes or to increased general acidosis. Oral administration of Witte-peptone resulted in increased amino acids, and indicated that the albumin products were irregularly digested by the liver and other organic cells. The indigo-carmine test showed pathological retardation of dye excretion, due, the authors concluded, to a diffuse hepatic dysfunction. An increased value of bilirubin was found in the serum and urobilin in the urine of patients with post-encephalitic residual phenomena. This was assumed to be the result of functional insufficiency of the hepatic cells.

Various tests, used in studying the vegetative nervous system, yielded contradictory and unequal results, owing to the imperfect and incomplete technique of investigating this sys-

tem. The authors concluded that the sequelae referable to the vegetative nervous system are conditioned to a considerable extent upon changes in the central nervous system and perhaps partially to disturbances of the peripheral (cerebrospinal and sympathetic) nervous system or of the functioning organs proper, or to changes in the endocrine glands.

The blood picture was also studied, without very conclusive findings.

The renal function was tested and pathological retardation of dye excretion was found.

The authors concluded that epidemic encephalitis is a disease of the entire organism, and not merely of the nervous system.

Cultivable Bacteria

Before reviewing the work done on cultivable bacteria, it may be well to discuss briefly spontaneous encephalitis in rabbits and also to give the few references that have been made to protozoa as possible etiologic agents.

Spontaneous Encephalitis In Rabbits

The rabbit has been used more extensively than any other animal in the experimental work on the etiology of epidemic encephalitis, along the lines both of cultivable bacteria and filtrable viruses.

Unfortunately the rabbit is subject to spontaneous infections that attack the central nervous system. Much has been written, especially in the past few years, in regard to these infections, and no attempt is made here completely to review the literature. Apparently Bull (B 425a) in 1917 was the first to call attention to the existance of spontaneous meningoencephalitis in rabbits. Later reports were published by Oliver (O 22-O 23), Twort and Archer (T 125a), Goodpasture

(G 100), and many others. Doerr and Zdansky (D 137) in 1923 called attention to the possibility of confusing these spontaneous lesions with lesions considered to be the result of experimental inoculations. The nature of these spontaneous lesions was thoroughly studied by Levaditi, Nicolau and Schoen (L 210, L 212, L 217, L 218, L 219, L 221, L 222) during the years 1923 and 1924. The lesions in the brain are characterized by a mononuclear meningitis, perivascular infiltrations and "encephalitic nodules" in and near which the parasites are found. The etiological agent is called by Levaditi, Encephalitozoon cuniculi. It was found to be a protozoon of group of Microsporidia. The virus was not filtrable. Lesions are found both in the brain and the kidney, and the disease is transmitted through spores in the urine. The parasite is found to be infectious for the mouse, the rat, and the dog, as well as for the rabbit. The disease in rabbits is essentially chronic and does not give rise, in the early stages at least, to any clinical evidence of its presence.

While this form of encephalitis in rabbits is apparently the most widespread and frequent, two other forms have been described.

Levaditi, Schoen and Sanchis-Bayarri (L 226, L 227, L 227a) in 1927 and 1928 described a form of spontaneous encephalitis in rabbits caused by the Toxoplasma cuniculi. This disease is apparently more acute than that caused by the Encephalitozoon cuniculi. The histological changes are quite different from those in other forms of experimental encephalitis—the alterations in the choroid plexus, in the aqueduct of Sylvius, and in the fourth ventricle being especially marked, with resulting hydrocephalus. Rabbits may be infected by inoculation in the anterior chamber of the eye. When this is done a keratitis and an exophthalmos develop, somewhat like those

described in a case of hydrocephalus in a child, cited by Levaditi (L 181.)

This form of encephalitis in rabbits has been described only by Levaditi and his co-workers, so far as we are able to ascertain. No statement has been made in regard to the prevalence of this disease.

A third form of spontaneous encephalitis, described by Rivers and Stewart (R 134b) in 1928, was caused by their filtrable virus III. It is stated that the lesions of the brain caused by this virus are at times quite similar to those caused by the herpes virus, but that these two viruses are not immunologically related.

Protozoa

Hilgerman (H 172, H 175) in 1920 reported that he found in blood smears (Giemsa stain) disc-like or pear-shaped objects resembling the Piroplasma parvum. These objects were especially numerous in the white blood cells obtained by liver puncture. They were also found in the spleen, bone-marrow and ventricular fluid.

In 1922, Hilgerman, Lauxer and Shaw (H 176) described the same protozoa in two additional cases and also a spirochete which the authors believed to be another stage in the development of the disc-shaped objects.

Dewes in 1921 (D 115) noted peculiar inclusion bodies in blood smears (Giemsa stain.) He wrote that these resembled somewhat the forms described by Hilgerman, but that he believed them to be Bagot's (Cabot's?) rings.

Wanner in 1926 (W 15) reported that he found by examination with the ultramicroscope actively mobile elements similar to spirochetes in the blood of patients, both in the acute and chronic stages of epidemic encephalitis. Guinea pigs

and white mice were inoculated intraperitoneally with this blood, with no effect. The author referred to the spirochete-like bodies described by Hilgerman.

Having concluded the brief discussion on spontaneous encephalitis in rabbits and the protozoa-like forms occurring in the blood of encephalitic patients, the work done on culitvable bacteria will be reviewed. Cultivable bacteria have been extensively studied as the possible etiologic agent of encephalitis.

Von Wiesner in 1917 (W 73) reported that he had found in the brains of fatal cases of epidemic encephalitis a diplostreptococcus, which both he and von Economo believed for a time to be the cause of epidemic encephalitis. An emulsion of the brain injected intracerebrally in a monkey produced symptoms in a few hours and death in forty-six hours. From the human brain and also from that of the monkey the diplostreptococcus was isolated in culture. It was Gram-positive, grew readily in bouillon containing glucose and later, on agar, and did not produce hemolysis. The brain emulsion injected intraperitoneally in a rabbit resulted in a hemorrhagic peritonitis.

Von Economo in 1920 (E 19) published a report confirmatory of von Wiesner's work, but in 1923 (E 25a) he stated that he no longer believed that the diplostreptococcus was the cause of epidemic encephalitis, but rather was a saprophytic invader.

Van Boeckel, Bessemans and Nelis (V 19) found the same organism in the brains of normal rabbits, especially when the material was not entirely fresh, and concluded that it was a secondary invader and not the etiologic agent.

Other apparently similar organisms were briefly described by several investigators as occurring in the brain or spinal fluid in cases of epidemic encephalitis. Numerous different

organisms in the blood, spinal fluid, or brain in cases of encephalitis have been found from time to time by smear or by culture. It does not seem of interest to refer to these observations unless careful cultural or experimental work was carried out.

Stafford in 1918 (S 301) found in two spinal fluids from a case of epidemic encephalitis a diplostreptococcus which did not hemolyze blood or produce greenish zones. It was not pathogenic for rabbits or guinea pigs.

Harbitz in 1920 (H 67) cultivated an apparently similar organism from the brain substance of two patients. No experimental work was reported.

Morse and Crump (M 386) in 1920 isolated a small Gram-positive coccus from the ventricular fluid, aspirated after death, of six patients. It closely resembled the organism described by von Wiesner. It was pathogenic for rabbits when injected subdurally, but not when injected intravenously. A sterile filtrate of a broth culture also produced death when injected subdurally in the rabbit.

Cantieri and Vegni (C 29) in 1920 reported the cultivation of a small Gram-postive diplococcus from the spinal fluid of four cases. The cultures were not pathogenic for rabbits or guinea pigs.

Pisani and Varisco (P 162), and Paleani (P 14) in 1920 isolated a small diplococcus, apparently Gram-amphophilic, from the blood or spinal fluid of several patients. The organisms were pathogenic for rabbits and guinea pigs.

House in 1920 (H 237) obtained a Gram-positive diplococcus from the brain of a fatal case eleven hours after death. It grew in ordinary media and produced a greenish zone on blood plates. It was not pathogenic for rabbits or guinea pigs. Rabbits injected at weekly intervals with a sus-

pension of the organism heated at 56° C. for half an hour developed immune bodies in the serum, by agglutination and opsonic tests.

Pico (P 146) inoculated rabbits intracerebrally with the spinal fluid of two cases of epidemic encephalitis. Some of the rabbits died and an emulsion of the brain injected subdurally in other rabbits produced death. From the brain of the second generation a Gram-positive diplostreptococcus was cultivated. Cultures of this organism also produced death when inoculated subdurally in rabbits and guinea pigs. The cultures from the spinal fluids and the first generation of rabbit brain were sterile. Filtered emulsion of the brain of the guinea pig and rabbit caused death after cerebral inoculation in the rabbit. The cultures of the brain were usually negative, but in one instance a diplostreptococcus was cultured from the brain of an animal inoculated wth a filtered emulsion.

Stern in 1920 (S 322) reported that in cases considered by him as typical, bacteriological studies were negative. Objects resembling a diplostreptococcus were sometimes seen in tissue, but he regarded them as artifacts.

In an atypical case with double influenzal pneumonia, an emulsion of the brain injected intraperitoneally in a guinea pig produced a purulent peritonitis from which an organism was isolated. This organism grew both aerobically and anaerobically, and appeared both as a streptococcus and as an encapsulated diplococcus.

Siegmund in 1920 (S 210) cultivated a diplostreptococcus in five instances from the brain or ventricular fluid. The organism was not pathogenic for guinea pigs, mice or rabbits. Siegmund believed that it was merely an associated organism and not the etiologic agent of the disease.

Zuccòla in 1921 (Z 19) stated that he cultured on ordinary

media a diplostreptococcus from spinal fluid. It was not pathogenic for laboratory animals.

Forte in 1922 (F 93) made essentially the same report.

Pappenheim in 1922 (P 28) reported the experiments of several workers in Russia. It was stated that Kulkow and Bjelikow cultured a pleomorphic diplostreptococcus from the spinal fluid and brain, either directly or after inoculation of the material in mice.

Dessy and Grapiolo in 1920 (D 105) isolated a Gram-positive diplostreptococcus from the spinal fluid of three patients. It was isolated by anaerobic methods and was at first very small, but later grew more readily and was larger. It was stated that its cultural and morphological characteristics lay between those of the diplococcus and the streptococcus. They believed it to be the same organism as that described by von Wiesner, although his streptococcus was isolated by aerobic culture. They suggested that this diplostreptococcus might be a "microbe de sortie" accompanying a filtrable virus—or that the filtrable virus described by other workers might be a filtrable form of this organism.

Reichert in 1920 (R 51) isolated a Gram-positive coccus, occurring usually in pairs, but also in chains, from the heart's blood and the basal ganglia of eight patients. It was isolated by anaerobic culture, but later grew aerobically. He believed it to be the same organism as that described by von Wiesner.

There is not much that can be said definitely in regard to these experiments with organisms identical with, or closely allied to, the diplostreptococcus of von Wiesner. One can only refer to the work of Van Boeckel, Bessemans and Nelis and the opinion of von Economo, already cited.

The experiments of Piazza in 1921 (P 134) may be briefly described. In one case out of thirty-six, the author isolated

a small motile bacillus, Gram-negative in young cultures, and Gram-positive in older growths. It grew aerobically and was pathogenic for rabbits and guinea pigs when injected into the veins or peritoneum. Lesions were found in the brains of the animals and the organism could be recovered from the brain. Piazza then inoculated emulsions of normal brain into the peritoneal cavity of animals of the same species and found that death occurred in from fifteen to twenty days, or in some instances in a few hours. This happened whether the brain emulsion was filtered or unfiltered. The phenomenon could be produced in series, and was due, the author believed, to a chemical action. He also injected cultures of a diplococcus from a case of penumonia, in rabbits and guinea pigs, and obtained the same results as other workers have reported with an organism supposedly of encephalitis. The histological lesions in the brain were the same and the organism could be recovered from the brain.

These two facts, the author believed, explained many of the reports in regard to the isolation of the specific agent of encephalitis.

An organism that is filtrable and is cultured anaerobically has been described by several investigators.

Bradford, Bashford and Wilson in 1919 (B 353, B 56, W 89) published articles describing a minute, filter-passing organism, cultured in Noguchi's tissue media, from the blood, spinal fluid, central nervous system and lymphatic glands of patients with encephalitis, and also from the brain of monkeys inoculated intracerebrally with an emulsion of the human brain. The organism was strictly anaerobic and remained viable for some months in 50 percent glycerin. A somewhat similar filter-passing organism was described for poliomyelitis, trench fever, influenza and nephritis.

No further work along these lines has been published by these authors, to our knowledge, and from personal conversation with bacteriologists in London, the impression was gained that some technical error had probably entered into these experiments.

Bastai in 1920 and 1921 (B 70, B 71, B 72, B 73) published four articles dealing with his researches. He inoculated cats subdurally with Berkefeld filtrates of brain emulsion from three fatal cases. A number of the cats developed symptoms and were killed on the sixth to the eighth day. The brains of the cats showed lesions. With the filtrate of the human brain and also of the cat, cultures were made anaerobically in media containing blood serum and fresh ascitic fluid. At the end of a week two organisms appeared—both cocci, but differing in size. The larger coccus grew aerobically in later generations. It did not hemolyze blood nor produce greenish pigment. The organism passed through the Berkefeld and Chamberland filters. It was pathogenic for rabbits, guinea pigs, rats and cats.

In his later papers he reported the successful inoculation of a monkey with spinal fluid of a new case, and also of a second monkey with the organism isolated in 1920. He stated that rabbits could be successfully inoculated through the nervous system, eyes or testicles, with his organism. In the cornea it produced varying degrees of keratitis. From the spinal fluid and blood, in 1921, he cultivated a diplococcus, which he said corresponded both to von Wiesner's diplostreptococcus and to the one that he had isolated in 1920. Experiments with this organism on animals gave negative results, which he claimed were due to a variation in the virulence of the infection.

Bastai's "virus," or more exactly culture, was examined by

Levaditi (L 178) and was pronounced by him to resemble the *Pasteurella.*

Schnabel (S 104) also examined specimens of the "virus-culture" of Bastai and found it to contain Gram-negative organisms. It evidently contained no herpes virus, as animals immune to the herpes virus reacted upon inoculation with this material in the same way as normal animals.

Maggiora, Mantovani and Tombolato in 1920 (M 33) found in blood cultures a small Gram-positive diplococcus, growing anaerobically. This organism was pathogenic for guinea pigs, and became more virulent when passed on in series. The patient's blood injected subcutaneously in guinea pigs caused death on the fifth to seventh day, with lesions in the brain. The human spinal fluid, though sterile by culture, injected subcutaneously in guinea pigs caused death on the seventh day, with a hemorrhagic process in the lung. A Gram-positive diplococcus was cultured from the guinea pig's blood.

Maggiore and Sindoni in 1920 and 1921 (M 35, M 36) reported the isolation in Tarozzi-Noguchi medium of a very small Gram-negative coccus from the blood and spinal fluid. The injection of the culture in the brain or veins of rabbits was followed by paralysis and death. Fresh rabbits inoculated with emulsions of the brain of infected rabbits developed the same symptoms, but more slowly. Cerebrospinal fluid at different stages of the disease always gave positive results when injected in rabbits.

The organism was resistant to 50 percent glycerin. It was believed by the authors to be identical with that described by Loewe, Strauss and Hirshfeld, by Thalhimer, and by Flexner and Noguchi, in poliomyelitis. Indeed the authors expressed the opinion that epidemic encephalitis and poliomyelitis are

identical, except for biological variations in the organism or differences in the persons affected.

Misasi in 1925 (M 316) stated that in ten cases of epidemic encephalitis in children, spinal fluid and blood cultures in Tarozzi-Noguchi medium showed an organism morphologically and culturally like that described by Maggiore and Sindoni.

Ottolenghi, D'Antona and Tonietti in 1920 (O 46) reported that the blood and spinal fluid was pathogenic for the guinea pig when injected subdurally, intracerebrally or intraperitoneally. Filtered nasal washings produced the same effect. In the guinea pig the disease was transmitted in series, for eight to twelve passages in some instances. Of 215 guinea pigs injected, only thirteen survived. The lesion in the brain was usually only hyperemia, rarely hemorrhage. A small pleomorphic, Gram-negative diplostreptococcus was isolated from the brain in anaerobic cultures in Noguchi's liquid, ascitic media. It was decidedly pathogenic for guinea pigs, less so for rabbits, and not at all so for cats.

Levaditi in 1925 (L 178) stated that this virus or culture was entirely different from his strain. He suggested that it might be a spontaneous, infectious disease of the guinea pig.

Ottolenghi and his associates in 1923-24 (O 47) said that Levaditi's work has refuted their experiments of 1920, and reported the isolation of a non-cultivable virus, which is described in the section on filtrable viruses.

Loewe, Strauss and Hirshfeld (L 297, L 298, L 299, L 300, L 301, L 302, L 303, S 379, S 381) in 1919 and 1920 made extensive studies by cultural methods and animal experimentation on the etiology of epidemic encephalitis. Their cultural work was successful only when Noguchi's kidney tissue, ascitic media, was used under anaerobic conditions.

Using this method cultures were obtained with 50 percent of spinal fluids; with 66 percent of Berkefeld filtrates of naso-pharyngeal washings; in seven out of ten instances with Berkefeld filtrates of naso-pharyngeal mucous membrane from fatal cases; with the human brain, especially in rapidly fatal cases (it was stated that difficulty was experienced in obtaining the culture from the human brain as it was often grossly contaminated, necessitating the use of filtrates); and with the blood in two instances. Positive cultures were obtained in 85 and 66 percent respectively from the brains of monkeys and rabbits that had been successfully inoculated.

The colonies appeared on the fifth to the seventh day. It was not possible to isolate the organism in solid medium, but it could be transferred to it in later generations. The organisms were minute, globular bodies, occuring singly, in pairs, chains and clusters. They varied in size, being smaller in young cultures, and appearing as larger degenerated forms in older growths. The staining was Gram-amphophilic, the younger forms and those in fluid media being Gram-positive; the older cultures, especially in solid media, being Gram-negative. The organsims were filter-passing, but the filter apparently held back many of them.

It was stated that monkeys were refractory to inoculations with the culture, but that more than 50 percent of rabbits inoculated with a filtrate of the culture in various generations succumbed with typical lesions in the brain. The incubation period varied from two to forty-two days. The authors did not believe that the successful inoculation with the cultures could be due to the carrying over of the original virus, as months elapsed between the original isolation of the organism and the time when some of the sub-cultures, up to the eleventh generation, were used for inoculation. After so many

sub-cultures there could be little possibility of enough of the original virus being present to have any effect. Moreover, the virus deteriorates rather rapidly at incubation temperature.

Attention was called to the morphological and cultural resemblance of this organism to the globoid bodies in poliomyelitis described by Flexner and Noguchi. It differed from these bodies, however, in its pathogenicity for rabbits and in the fact that it was easily cultivated from spinal fluids.

In carrying out the cultivated studies controls were made using nasal washings, nasopharyngeal mucous membrane, spinal fluid and brain emulsion from patients suffering from other diseases—all with negative results.

Experiments were also carried out by inoculating animals directly with material from human cases—brain emulsion, spinal fluid and filtrate of nasopharyngeal washings and of nasopharyngeal mucous membrane.

In their first experiment they inoculated a monkey (*Macacus rhesus*) subdurally and intraperitoneally with a saline emulsion of a human brain. On the following day the animal became ill and two days later it was killed. The brain showed lesions—multiple cortical and subcortical punctate hemorrhages, perivascular round-celled infiltration, and a certain degree of meningitis. An emulsion of the brain was injected intraperitoneally and intracerebrally in a second monkey, which became paralyzed on the sixth day and was killed seven days later. This brain also showed lesions and, after being kept in cold storage for two months in 50 percent glycerin, was inoculated in two rabbits. Both rabbits died and in the brain of one typical lesions were found.

A purulent nasal discharge from a patient was passed through a Berkefeld filter and (after being proved sterile by

culturing on ordinary media) was inoculated intracranially and intracerebrally in a *Macacus*. The monkey developed symptoms on the eighth day, but recovered ten days later. In two months the animal was again inoculated intracranially with a highly virulent filtrate of nasal mucous membrane and showed no reaction. It was assumed that the monkey had acquired an immunity.

It was found that the rabbit was much more susceptible to inoculations than the monkey. A filtrate of human brain from three cases was inoculated in twelve rabbits. Four died, three of which showed lesions in the brain. An emulsion of the human brains of two cases was injected in six rabbits, four of which died, two showing lesions in the brain. A filtrate from the nasopharyngeal mucous membrane of a fatal case was injected intracranially in a rabbit, with the production of typical lesions. This virus was transmitted through six rabbits, then to a monkey, and then back to two generations of rabbits, using each time a filtrate of brain emlusion for inoculation.

The injection of filtered nasopharyngeal washings intracranially in rabbits gave positive results in 78 percent of cases and was suggested as a means of diagnosis in doubtful cases of epidemic encephalitis.

The inoculation of spinal fluid in rabbits gave positive results in 61 percent of cases and was also suggested as a means of diagnosis.

Control inoculations were carried out using similar material from patients suffering with other diseases, with negative results.

Thalhimer in 1921 (T 34), reported the isolation of somewhat similar organisms by the same cultural technique, from the spinal fluid of two cases, from the filtrate of the brains

of four cases, and from about 80 percent of the filtrates of the brains of rabbits successfully inoculated with the original material. These organisms were carried through several generations. They passed through the Mandler filter. Positive results were obtained by the injection of the culture intracranially in rabbits and the culture was recovered from the brain filtrate of the rabbits.

When rabbits were inoculated intracranially with human material—brain filtrate, spinal fluids, or nasophryngeal washings—positive results were obtained in a large percentage of the cases. The animals usually died in from two to eight weeks. In the brains of some of the animals that died no lesions were found, even though the virus was present, as shown by positive cultures or by transmissions to other animals.

Viruses of different origins were injected intracerebrally in fifty-two guinea pigs, of which thirty-five died, with twenty-one showing characteristic lesions.

In 1922-23 Thalhimer (T 35) reviewed his earlier work and stated that, of the animals injected in groups, 50 to 60 percent as a rule were affected. He also added that the organism had been recovered from the spinal fluid of animals injected with the virus.

Control experiments, both in the cultural work and in the animal inoculations, were carried out, using the same kind of material from patients with other diseases. The results with these materials were negative.

Commenting on the experiments reported by Loewe, Strauss and Hirshfeld, Flexner (F 64) stated that with different materials—brain emulsion, filtered nasopharyngeal washings and filtered extracts of nasopharyngeal mucosa from more than forty patients, and more than one hundred spinal fluids—

inoculations were made in both monkeys and rabbits in an attempt to transmit epidemic encephalitis to animals. In no instance was the attempt successful. Occasionally a meningitis or an encephalitis followed the intracerebral inoculation, but careful study proved it to be due to an accidental or intercurrent infection. In one instance a herpes virus was isolated from the spinal fluid of a patient suffering from neurosyphilis. This is described under the section dealing with filtrable viruses. Flexner also called attention to the fact that McCartney, after a careful study of more than three hundred rabbits at the Rockefeller Institute, found the lesions of spontaneous encephalitis in about half the number. It was pointed out that the percentage was about the same as that of the positive results reported by Loewe, Strauss and Hirshfeld. Moreover, in Flexner's opinion, the lesions in spontaneous encephalitis resembled quite closely those in the reports and photographs published by Loewe and his associates.

McCartney in 1926 (M 200) published a report of his attempt to repeat the cultural work done by Loewe, Strauss and Hirshfeld. He stated that the tubes of uninoculated media showed the same clouding as those inoculated with material from cases of encephalitis. This he believed was due to a precipitation caused by an alteration of the hydrogen-ion concentration. The "globoid bodies" were in the control tubes as well as in those that had been inoculated.

Levaditi in 1925 (L 178) stated that he worked with Thalhimer's virus and found it to be the "encephalitozoon cuniculi." He believed that Loewe, Strauss and Hirshfeld were working with the same infection.

Evans and Freeman in 1926 (E 81) reported the isolation of a pleomorphic streptococcus from the heart blood and mesencephalon of a patient dying in the third exacerbation of

epidemic encephalitis, six years after the first attack. These materials were planted in the meat broth medium described by Evans. "Anaerobic cultures are grown in a meat medium prepared like ordinary beef infusion broth; but instead of discarding the meat from which the broth is made, the ground meat particles are placed in the tubes to a depth of about one inch. After the medium has been inoculated, a cap of sterile melted vaseline is added." Anaerobic cultures were obtained which were injected intracerebrally in rabbits. From the brains of the rabbits, which reacted to the inoculation, cultures were obtained which grew aerobically on vitamine agar as well as in the anaerobic meat broth medium. A third strain was obtained by inoculating rabbits intracerebrally with nasal washings taken eight days before death. From the rabbit brain a culture was obtained which grew both on vitamine agar and in the meat broth medium. These three strains resembled each other culturally, morphologically, and in their pathogenicity for rabbits. The strains from the nasal washings and from the heart blood were shown to be identical by cross protection tests.

The organism was Gram-amphophilic, produced a slight hemolysis and a greenish tinge on blood agar, grew in ordinary media in subcultures, and was highly pleomorphic. It varied greatly in size, sometimes being so small as to pass through a filter and at other times being described as a "giant" coccus. It also appeared as a diphtheroid and as a spore-forming rod, with minute filtrable coccoid bodies appearing as buds on the walls of the rod. It was stated that "this report will be limited to observations on the streptococcus form of the organism—the form in which the virulence is highest and most stable."

Cultures of the streptococcus in the meat broth medium,

kept at 4° C., maintained their virulence for five months. Organisms in the brain of a rabbit, preserved in glycerin at 4° C., lost their virulence by the forty-eighth day.

The streptococcus was highly virulent for the rabbit, producing death in from five and three-quarters to eighteen hours, when injected intracerebrally. There was more variation in the virulence when the organism was injected intravenously in rabbits. After this method of inoculation the streptococcus could be recovered from the brain. Four monkeys were inoculated with the culture, three of which died. The fourth had been inoculated six months before with a culture of a coccus obtained from a case of poliomyelitis and it was assumed that this monkey had in this way been immunized.

It was found possible to immunize rabbits to large intracerebral inoculations by three or four intravenous or intraperitoneal inoculations.

The rapidity of death following the intracerebral injections of the culture in rabbits suggested to the authors the action of a toxin. This was disproved by inoculating a filtrate of the culture, which proved harmless. The authors stated that filtrates of emulsions of the human brain and of rabbits' brains also gave negative results when inoculated intracerebrally. The authors continued: "By planting filtrates in meat medium, however, virulent cultures have been obtained and they have shown that the organism is in a filterable form in the human brain, in the rabbit brain, and in cultures."

The study was summarized as follows: "A pleomorphic streptococcus, highly virulent for rabbits when inoculated intracerebrally, was obtained from the nasal washings, heart blood, and mesencephalon of a case of epidemic encephalitis.

"In so far as the comparative tests have been made, this streptococcus agreed with the streptococci obtained from cases

of epidemic encephalitis by von Wiesner and by Rosenow. Apparently several other investigators have cultivated the same organism in their studies of the disease.

"When inoculated intravenously into rabbits the streptococcus shows a tendency to elective localization in the brain.

"In rabbits and in monkeys it produces nervous symptoms which in some cases simulate the disease in man.

"Rabbits inoculated with this streptococcus show no inflammatory lesions outside of the central nervous system. The meninges are heavily infiltrated with lymphocytes and leucocytes, the inflammation spreads to the cerebral substance by direct extension and along the small vessels, penetrating into the brain. There are severe parenchymatous degenerative changes in the nervous tissue and reaction of the neuroglia. The sheaths of the blood vessels are found infiltrated by lymphocytes. The reaction is sometimes most marked in the mesencephalon.

"In monkeys there is noted a greater tendency toward leucocytic reaction, and in two instances large areas of hemorrhagic inflammation in the basal ganglia were noted."

Freeman in 1926 (F 120) reported that a similar organism had been isolated from the spinal fluid of two cases in the chronic stage of epidemic encephalitis and in the blood and spinal fluid of two cases in the acute stage.

In 1927 Freeman (F 121) stated that the same organism was isolated by Miss Evans from the blood during life and from the brain after death of a patient suffering from encephalitis and brain tumor. In another publication in 1927 Freeman (F 123) reviewed the earlier work and reported the isolation of the same culture from additional cases, some of them being cases of "idiopathic" paralysis agitans in which there was no history of encephalitis.

In 1927 Evans (E 80) obtained six strains of virus from various investigators, one of which had been isolated from a case of epidemic encephalitis, four from herpetic vesicles, and one, the virus "Beckley," from the spinal fluid of a syphilitic patient.

All of these viruses produced the typical herpetic syndrome when injected intracerebrally in rabbits, death occurring in from three to eight days after inoculation. A careful examination of smears from the brains of these rabbits revealed a few cocci, and cultures were obtained by inoculating the brain of the rabbit, in the meat broth medium, used for the cultivation of streptococci with materials from cases of epidemic encephalitis. By this method organisms were cultivated from all six of the viruses. These organisms closely resembled those already described, which had been cultured from cases of encephalitis. They assumed both the streptococcus and the spore-producing rod forms. It was stated that in some instances the cultures were obtained directly from the virus preserved in glycerin, as it was received at the laboratory. The cultures were sometimes grown from filtrates of the brain emulsion. These six strains differed in virulence, but all were less pathogenic than the strains isolated from cases of epidemic encephalitis.

This group of experiments, and especially those reported in the last publication of Evans, led Olitsky and Long (O 21a) in 1928 to study the relationship between streptococci and herpes virus encephalitis. For their experiment they used two strains of virus that had been used by Evans, and also Levaditi's strain C. The media were prepared according to the directions given by Evans. Cultures were made both in the meat broth medium and on blood agar plates. The following materials were used: herpes virus-infected rabbit brains

kept from eight to 113 days in 50 percent glycerol; fresh virus-infected brains of rabbits and guinea pigs; fresh brains of guinea pigs inoculated intracerebrally with non-infective material, as a control. The blood agar plates remained sterile in all instances. From the meat broth cultures a *Staphylococcus aureus* was isolated from one tube containing glycerolated material; a streptococcus and a large coccus from fresh virus-infected brains of rabbits; two strains of streptococci from fresh virus-infected guinea pig brain; two strains of streptococci from the control guinea pig brain.

An experiment was tried concerning the possibility of obtaining organisms from ground meat particles and dextrose broth—the process of grinding being carried out with the same precautions against contamination that were used in grinding the brains of the animals in preparing the emulsion.

From tubes of meat medium inoculated with the "ground" meat particles and the ground dextrose broth, streptococci, giant cocci, diphtheroids, spore-producing rods and other organisms were isolated. The blood agar plates inoculated at the same time were sterile. It was believed that the organisms were contaminants introduced through the process of grinding. The streptococci corresponded culturally with those described by Evans. Four of eight strains of these streptococci tested, proved pathogenic for rabbits, producing a purulent meningoencephalitis.

No cross immunity was demonstrated between the streptococci and the viruses.

It was shown that the virus was more resistant to 50 percent glycerol than was the streptococcus. The authors stated that: "From all this it can be concluded that streptococci are not the visible forms of herpes virus, nor do they produce in

rabbits effects like those induced in the brain and cornea by the herpes virus."

Zinsser in 1928 (Z 14a) wrote as follows: "The problem in regard to the work of Evans and Freeman is of much the same order as that regarding the streptococci claimed by Rosenow in poliomyelitis; and a strain of virus placed at my disposal by Dr. Evans has been found, in many passages in rabbits to be identical pathogenically and immunologically with strains of herpes virus. A large number of cultivations made by my associates, Drs. Tang and Yu, from rabbits infected with this strain have occasionally yielded streptococci, but the organisms so obtained have repeatedly failed to produce a disease in other rabbits anything like that produced by the brain substance itself. If one assumes the possibility of a mutation of an organism into a filtrable virus form, and together with that, a modification in pathogenic properties during the course of this mutation, one could accept as a solution of the problem not only the work of Evans and Freeman, but that of others who have cultivated bacteria. It is still a far cry experimentally to such a premise, however, and logic forces one at the present time to reject the bacterial causation of this disease on the following grounds:

"1. The clinical symptoms and speed of death of inoculated animals not only is different from that of the human disease, but differs materially from that following the inoculation of the herpes virus into the same species of animals.

"2. The pathologic changes in encephalitis in man are fundamentally different from those produced by any of the known forms of bacteria.

"3. The nature of immunity, as far as is known in encephalitis, and certainly in filtrable virus diseases generally, and

herpes especially, is different, both biologically and serologically, from that familiar in streptococci infections.

"4. In addition to this, all those who have seriously claimed bacterial etiology for this disease have assumed an interrelationship between the bacteria and a filtrable stage, in which case it would be necessary to stretch this mutation not only into one of form and infectious properties, but into one affecting all the basic biologic attributes of the agent; for—unlike bacteria—the filtrable virus can be preserved in glycerin indefinitely, degenerates easily in salt solution, and is much less resistant to heat, chemicals and most other deleterious agencies than are bacteria.

"Altogther, then, a poor case is made out for bacterial causation, which must be thrown out entirely, except on the slim possibility of actual mutation, for which the evidence is entirely too unreliable at the present time to permit anything more than a purely speculative view."

Rosenow has doubtless devoted more time to the study of the etiology of epidemic encephalitis from a bacteriologic standpoint than has any other investigator. These experiments have been conducted along the same lines on which Rosenow has worked for many years with multiple sclerosis, transverse myelitis, neuralgia, multiple neuritis, poliomyelitis and other diseases. Rosenow has concluded that streptococci with specific or elective localizing power play an important part in the etiology of these diseases. With this background Rosenow began his investigations of the etiology of epidemic encephalitis.

His first paper was published in 1921 (R 247a) and dealt with the isolation of a pleomorphic green-producing streptococcus from material from tonsils, nasopharynx and pyorrhea pockets of three cases of epidemic hiccup—a condi-

tion the cause of which Rosenow believed to be similar to the cause of epidemic encephalitis. With the material from the patients and also with the culture, inoculations in rabbits and monkeys produced spasms of the diaphragm. The inoculation period varied from one to seven days, depending on the size of the dose and its virulence. Many of the animals recovered. When the animals died or were chloroformed, the same organism was found in the brain in active lesions, and also at times in the spinal fluid. It was difficult to demonstrate the organism in the brain in chronic lesions and they were never found after recovery.

After animal passage the specificity of the organism seemed to change. The hiccup decreased and lethargy and symptoms of epidemic encephalitis developed.

In 1922 Rosenow (R 248) reported his experiments with a green-producing streptococcus isolated from a case of epidemic encephalitis from the following sources: infected teeth; filtrate of the nasal washings; the blood during life; an emulsion of the base of the brain; and a filtrate of the brain emulsion. Stained sections of the base of the brain revealed pleomorphic diplococci in or near the lesions. The organisms were isolated in glucose-brain-broth. For inoculations in animals the cultures were transplanted from three to six times in twenty-four hours for many generations. With such a culture it was possible to infect rabbits by intracerebral inoculations and also by packing the nose with gauze soaked in the culture.

Twenty-six rabbits and one monkey were injected intracerebrally with brain emulsion, with filtrate of the brain emulsion, filtrate of nasal washings and other materials, with negative results. This Rosenow interpreted as proving that a filtrable virus was not present and that the relatively few

streptococci in these materials were not sufficient to overcome the resistance of the animals.

It was stated that the streptococcus from the infected teeth, from the blood and from the base of the brain were the same with respect to morphology, cultural characteristics and immunologic properties. The strains isolated from the brain and from one infected tooth were neurotropic. The organisms, when freshly isolated, were agglutinated by anti-poliomyelitis and anti-encephalitis sera. After prolonged cultivation they were agglutinated also by influenza serum, anti-pneumococcus serum and normal horse serum.

In 1923 four papers were published. In one of these, Rosenow (R 249), dealing with the serum treatment of epidemic encephalitis, stated his belief that he was working with the same organism as that described by Loewe, Strauss and Hirshfeld, but that he was dealing with a larger aerobic form of the streptococcus. He found this strain closely related to his poliomyelitis strain.

A more extensive study of epidemic hiccup was also published in two parts (R 250). The results of the study of eight cases were reported in detail. In general these were much the same as those reported with the first three cases of epidemic hiccup. In the second part of the report, however, attention was called to the fact that rabbits developed the same symptoms when inoculated intracerebrally with dead bacteria as when inoculated with the filtrates of cultures. Cultures from the brains of these animals did not yield streptococci nor did emulsion of their brains produce symptoms when injected intracerebrally in normal rabbits. This was interpreted to prove that the symptoms were not due to a filtrable virus. Sections of the brain showed diplococci after injection with the dead cultures, but not after injection with the filtrate. Rosenow

stated: "Hence, the specific localizing power with the production of this highly characteristic syndrome would seem to be due to a chemical substance produced either by the streptococcus or during the reaction incited in the host."

In a third paper published in 1923 (R 251), attention was called to the similarity between the streptococcus isolated from cases of epidemic encephalitis and that isolated from cases of influenza. A careful study was made of a streptococcus isolated from the throat of a patient suffering from epidemic encephalitis, who had recently recovered from an attack of influenza. Three weeks after the onset of the encephalitis his daughter developed poliomyelitis and died from respiratory paralysis.

The organism was isolated from the brain of rabbits inoculated intracerebrally with washings from the patient's throat. It was agglutinated by Rosenow's anti-poliomyelitis serum, and also by the patient's own serum during convalescence. Rabbits, monkeys, mice and guinea pigs were inoculated with the organism by various routes. Following animal passage, it seemed to lose its neurotropic power and to gain a pneumotropic tendency. Coincidentally with this change, its agglutination reaction changed. It was agglutinated less successfully by the anti-poliomyelitis and anti-encephalitis serum and better by pneumococcus serum, especially Type 3.

Rosenow concluded: "According to the results of these experiments, the same streptococcus may give rise to these widely different diseases, depending on whether it is in the virulent or pneumotropic phase, when influenza would result, or in the less virulent, but neurotropic phase, when poliomyelitis and allied conditions would be prone to develop."

In the fourth article in 1923, by Rosenow and Jackson (R 258), it was stated that in stained sections of the brains of

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twenty-one cases of epidemic encephalitis, cocci or diplococci were demonstrated in or near the lesions. The organisms were more numerous in cases of short duration. Their morphology showed a wide variation. No other bacteria were found.

In 1924 Rosenow (R 252) isolated a similar streptococcus from seven cases of respiratory arrhythmia following epidemic encephalitis. The streptococcus was usually isolated by injecting rabbits intracerebrally with nasopharyngeal washings or with the pus from tonsils. Cultures of this organism produced respiratory disturbances when injected intracerebrally in rabbits and monkeys.

As in previous experiments, the streptococcus was frequently isolated from the spinal fluid, as well as from the brain of animals which died in the acute stage of infection after intracerebral inoculations.

The organisms resembled those isolated in previous experiments and five of the strains were agglutinated by Rosenow's anti-poliomyelitis and anti-encephalitis sera. This organism also after rapid animal passage developed the power to produce hemorrhagic broncho-pneumonia in the guinea pig.

In a second article in 1924 (R 255), the earlier work was reviewed, and it was stated that the streptococcus was isolated from four cases of spasmodic torticollis, three cases of chorea, five cases of Parkinsonian encephalitis, two cases of myoclonic encephalitis and two cases of respiratory arrhythmia. These organisms were much alike in morphology, cultural characteristics and virulence, but corresponded in their localization and the type of symptoms produced to the sources from which they were derived. Endocarditis and myocardial lesions followed the intracerebral injection of the strains from cases of chorea, and the organism was isolated from the blood in 31

percent of the animals injected. These results were not obtained after the injection of strains from the other sources. The localization of the lesions in the brain also varied with the source of the strain: the basal ganglia being most frequently involved with strains from Parkinsonian encephalitis and spasmodic torticollis; the cortical and subcortical regions, with strains from myoclonic encephalitis and chorea; and the anterior part of the medulla, with strains from the cases of respiratory arrhythmia.

A third paper (R 253) was of the nature of a general review. It was stated that when rabbits were inoculated with washings from the nasopharynx, pus from the tonsils, or cultures from these sources, there was a mortality of 74 percent with strains from cases of epidemic encephalitis; 40 percent with strains from contacts with these cases; and 23 percent with strains from normal controls or patients suffering from other diseases.

A very comprehensive study of eighty-one cases was also published in 1924 (R 254). Of these eighty-one cases, thirteen were of the lethargic type; fifteen of the Parkinsonian type; seventeen of the myoclonic type; five of the meningitic type; and thirty-one were unclassified.

Cultures were also made from eleven contacts and from seventy-seven normal controls.

The cultures were obtained, by the methods already described, from foci of infection, nasopharynx, tonsils and teeth; from the brain and medulla of fatal cases; from the spinal fluid of three of the five meningitic cases; and from four other cases.

The characteristics of the organisms were described. The fermentation reactions were variable. The smaller forms, especially those growing in old anaerobic cultures, were filterpassing.

Most of the encephalitic strains were agglutinated both by Rosenow's anti-poliomyelitis serum and anti-encephalitis serum, and by the serum from patients suffering from encephalitis. The serum of normal persons had little or no agglutinating power. The encephalitis and the poliomyelitis strains were immunologically similar.

Cultures of these streptococci, material from the patients, and also filtrates of the material and of cultures were inoculated in rabbits, monkeys, guinea pigs and mice. While intracerebral inoculations gave the highest percentage of positive results, localization in the brain followed intravenous and intraperitoneal injections in a varying percentage of instances. Inoculations of animals with morphologically similar cultures from control cases yielded a very small number of positive results.

The symptoms developing in the animals are described as being very similar to the symptoms of the patients from whom the cultures or material were obtained.

The microscopic lesions in the brain were described as follows:

"The microscopic changes varied greatly according to the duration of the experiment, the size of the dose, and the virulence of the strain injected. Leukocytic and round-cell infiltration of the pia was most marked surrounding blood vessels in the sulci. Focal hemorrhages in the brain and medulla, often perivascular, associated with a variable degree of leukocytic and round-cell infiltration, and similar areas of infiltration unassociated with hemorrhage were the usual findings in the animals that succumbed or were anesthetized in from 1 to 5 days after injection. The organism was readily demonstrated in these lesions. As the duration of

the experiment became longer, hemorrhages were less common, the leukocytes were displaced by round cells, and the diplococci became fewer and more irregular in size, large and small forms often occurring side by side and sometimes in the same chain. The proportion of leukocytes and round cells varied greatly according to the location of the lesion, the former often predominating in the pia and the latter in the perivascular spaces in the depths of the brain substance."

Rosenow again suggested the possibility that he was dealing with a larger aerobic form of the smaller anaerobic organism isolated by Loewe and Strauss, Thalhimer and others.

In 1926 two articles were published (R 256 and R 256a) dealing more particularly with the relation of epidemic hiccup to epidemic encephalitis. In the first paper (R 256) twenty additional cases of epidemic hiccup were studied. Cultures were obtained, similar to those already described, and animals were inoculated, 76 percent of which developed muscular spasms.

The work of Evans was cited as corroborating Rosenow's experiments with the streptococcus as the etiologic agent of epidemic encephalitis.

This streptococcus was agglutinated by the anti-poliomyelitis and anti-encephalitis sera and also by the sera of convalescent patients.

Rosenow concluded that a close relationship between epidemic hiccup and epidemic encephalitis was indicated by his experiments.

The second paper (R 256a) dealt with experiments on five cases of neuromyelo-encephalitis occurring during and following an epidemic of hiccup.

Cultures of a streptococcus were obtained from the nasopharynx or tonsils of these patients by the same technique that

was used in epidemic encephalitis and epidemic hiccup. In these experiments dogs as well as rabbits were used for inoculation, both with suspensions of the swabbings from the nasopharynx and tonsils and with the cultures. The animals were reported as developing symptoms closely resembling those in the patients from whom the materials were taken. Four dogs, injected with material from two patients with nausea and vomiting, manifested these symptoms.

All five of these patients suffered from neuritis. Hemorrhagic lesions of the nerves occurred in eight of twenty-one rabbits injected intracerebrally and in ten of seventeen injected intravenously. A very small percentage of rabbits injected intracerebrally with material from cases of epidemic hiccup developed hemorrhages in the peripheral nerves or nerve roots.

Lesions containing the streptococcus were found in a few instances in the stomach or colon of rabbits injected intracerebrally, a fact which Rosenow concluded was in harmony with the occurrence of gastro-intestinal symptoms during the epidemic.

These organisms were culturally and morphologically like those isolated from epidemic encephalitis and epidemic hiccup. They were agglutinated by Rosenow's polio-encephalitis serum.

The conclusion was:

"Since changes in localizing power were noted in some of these strains following artificial cultivation and animal passage, the conclusion seems warranted that the changes in the character of the epidemic, from hiccup to other manifestations of a neuromyelo-encephalitis, were due to change in the tropism or localizing power of the streptococcus isolated."

In 1928 Rosenow (R 257) published a paper in which his earlier work was reviewed and studies of additional cases were related. Attention was called to the work of Evans and

Freeman, which the author stated paralleled his own in important respects. Reference was made to viruses and to Evans' report of the isolation of streptococci with neurotropic power from such viruses.

In Rosenow's new series of cases, five were cases of epidemic hiccup, nine of epidemic encephalitis, six of myoclonic encephalitis, thirty of Parkinsonian encephalitis, five of respiratory arrhythmia, sixteen of spasmodic torticollis and seventeen of chorea. Rabbits were injected intracerebrally with material from the nasopharynx, tonsils or teeth, or with primary cultures in dextrose brain broth. It was stated that evidence of focal infection, especially in the teeth, was found in most of the cases. It was also stated that "the incidence in the animals of the symptoms most characteristic of the different groups was often strikingly high and in some instances simulated the condition in the patient so closely as to make one feel certain that the material injected must have contained the etiologic agent."

From the inoculated rabbits, the green-producing streptococcus was isolated in 67 percent of cases from the brain, and in 34 percent of cases from the blood. In a few instances hemolytic streptococci, staphylococci and other organisms were obtained.

It was reported that the microscopic changes in the brain were quite similar in the animals inoculated with strains from the different groups, differing in site rather than in character. It was at times not possible to correlate the lesions found after death with the symptoms that had been observed.

Rosenow concluded:

"Another series of experiments with animals performed with material from foci of infection or throats of patients suffering from various diseases of the nervous system is re-

ported. The results corroborate and extend the former observations. The streptococci isolated in each of these conditions, while much alike, possess very different localizing and symptom-producing power when injected intracerebrally into animals and readily lose this property, especially on aerobic cultivation. The specific localizing power of the streptococcus, it would seem, is dependent, in part at least, on the production of a toxin or poison that has specific effects. Lethargy, hyperpnea with marked arrhythmia, and the symptoms characteristic of spasmodic torticollis have been produced with filtrates from nasopharyngeal washings, filtrates of suspensions of the pus from tonsils and filtrates of old cultures of the specific streptococcus in each of these conditions. The fact that the streptococcus, having specific effects and the power to produce a specific poison, has been isolated long after onset indicates that the late manifestations of encephalitis and allied conditions are not sequels but are due to the activity of the causative organisms or its toxin, as has been especially emphasized by Freeman.

"It is of course realized that the results obtained are not absolute proof of the etiologic relationship of the streptococcus isolated. None of the patients in this group died, and hence search for the organism in lesions could not be made. Agglutination experiments, especially with the serum of patients and the suspension of the suspected organism while sometimes positive in low dilutions, were not so regularly enough to be convincing. The benefits which follow the removal of foci proved to contain the organism and the use of autogenous vaccines, while often striking, do not occur regularly enough to prove etiologic relationship.

"In consideration of all the facts, especially the extremely specific effects obtained in some cases with the streptococcus

isolated, and its specific toxin or poison-producing power reported herewith, the tentative conclusion seems warranted that this organism has etiologic significance in each of the diseases studied."

Filtrable Viruses

The researches on the etiology of epidemic encephalitis have dealt more with filtrable viruses than with any other agent.

Instead of arranging this work chronologically it seems better to consider these viruses in three groups:

A. Those that have been quite definitely disproved, by later work, as possible etiologic agents.

B. Those in regard to which too little work has been done to permit of any definite conclusions.

C. Those viruses closely allied to, or identical with, the herpes virus, which have been isolated in several instances from the brain or spinal fluid in cases of epidemic encephalitis.

Group A.—Since 1921 Kling and his co-workers, Davide and Liljenquist, have contributed numerous articles on the etiology of epidemic encephalitis. In 1921 (K 67, K 68, K 69, K 70, K 81) they reported that they had reproduced epidemic encephalitis in rabbits by inoculation with brain substance, cerebro-spinal fluid, nasopharyngeal secretions and washings of the intestinal tract. The disease in rabbits was usually chronic, often lasting from forty to fifty days or longer, and it was also stated that the animals sometimes had no symptoms and yet showed the pathologic lesions. The injections were made in the brain and also in the peritoneal cavity. The virus was carried on in series. Attempts to reproduce the disease in monkeys were unsuccessful.

It was stated that the virus was filtrable, was not cultivable, and was preserved in glycerin.

In 1922 (K 65, K 71, K 72, K 73, K 74, K 75, K 76) they reported further studies. It was stated that ten strains of virus had been isolated from cases of epidemic encephalitis—four of cerebral, four of nasopharyngeal, and two of intestinal, origin. They again stressed the chronicity of the disease in rabbits, stating that it lasted from one to seven months and that pathologic lesions were found in rabbits that had failed to show symptoms. They stated that they had found no cases of spontaneous encephalitis in the new rabbits which they examined. They were unable to reproduce the disease in apes, thirty inoculations having been made with negative results.

The authors were interested in the experiments published by Levaditi, Doerr and Schnabel, and others and suggested the possibility that the viruses isloated by these workers were forms of herpes virus. To study this point further they isolated two strains of herpes virus and studied them in comparison with their own strains of virus. They found that the herpes virus was pathogenic for rabbits, guinea pigs and white mice, while theirs was pathogenic only for rabbits; that the herpes virus produced an acute encephalitis, while that produced by theirs was chronic; that corneal inoculation with the herpes virus produced a kerato-conjunctivitis, followed by an acute encephalitis, while with theirs there was a thickening of the corneal epithelium, followed by a chronic encephalitis; that the pathologic lesions in the brain were different; that there was no cross-immunity between the two kinds of viruses; and that both were filtrable and resistant to glycerin. They further stated that the serum of convalescent patients of epidemic encephalitis neutralized their virus to a considerable degree, while it did not do this with the herpes virus.

They also reported the isolation of their virus by inoculating rabbits with spinal fluids of patients in the chronic stage of the disease.

In 1923 (K 66, K 77) the articles published repeated the statements previously made. The additional fact was added that while Levaditi's virus and the herpes virus were destroyed at 55° C., the Swedish virus resisted heat at 65° C.

In 1924 Kling, Davide and Liljenquist published three papers (K 78, K 79, K 80) in which they stated that they had taken great care to rule out the possibility of spontaneous encephalitis in rabbits as a factor in their experimental work. They reported that they had isolated twenty-three stocks of virus from cases of epidemic encephalitis.

The statements made by Levaditi, and Doerr and Zdansky as to the identity of the Swedish "virus" with spontaneous encephalitis were discussed at length.

Meantime they had obtained a specimen of Levaditi's virus and had shown its close relationship in symptomatology, histological changes and immunity reactions to the virus of herpes.

Kling asked why, if this herpetic-like virus is the cause of encephalitis, he had never isolated it in the sixty-eight cases he had examined. The answer is indeed diffcult; however, as will be stated later in more detail, Doerr and Zdansky did isolate a herpes strain from a brain sent them by Kling.

In 1927 Kling (K 66b) published a paper in which he expressed somewhat modified views, though apparently not fully accepting the opinions of several investigators that the Swedish "virus" is identical with spontaneous encephalitis in rabbits. In this article it is stated that the Swedish virus is non-filtrable, thereby contradicting the statement made in several earlier papers that it is filtrable.

Meantime Levaditi, Nicolau and Schoen in 1923 and 1924 (L 210, L 212, L 217, L 218, L 219, L 221, L 222) had examined the "virus" of Kling and had pronounced it to be the same as spontaneous encephalitis in rabbits.

Doerr and Zdansky also in 1923 (D 137) had stated that the granulomas in the brains of some rabbits innoculated with Kling's virus were probably due to a parasitic infection of the animals entirely different from human encephalitis. In 1924 (D 140) they repeated these statements. Schnabel in 1924 (S 104) corroborated this.

Auriat and Flye Sainte Marie (A 164) in 1924 reported the corneal inoculation of a rabbit with the spinal fluid of a Parkinsonian case. No corneal reaction developed and the animal was sacrificed four months later. Histological lesions were found in the brain, including nodules. It is, therefore, probable that the lesion was that of spontaneous encephalitis.

In 1925 and 1926 F. H. Lewy, F. M. Lewy, Fränkel, Kuttner and Kantorowicz (L 246, L 247, L 248, L 249), after making comparative tests with Kling's virus and the virus of "street distemper" in dogs, concluded that there was a cross immunity between these two viruses. They stated that Kling's virus was not filtrable, but that the virus of distemper was filtrable. Levaditi's herpes-encephalitis virus was found filtrable and there was no cross immunity either with Kling's virus or with the virus of distemper.

Koritschoner in 1923 and in 1925 (K 100, K 101) reported the isolation of viruses from two fatal cases of encephalitis or myelo-encephalitis in which the subjects had been bitten by rabid dogs about six weeks prior to the onset of the acute illness. The viruses were first isolated by subdural injections in rabbits of an emulsion of the brain. Later the virus was successfully injected in dogs. Luger and Lauda

(L 347) examined this strain and considered it to belong to the herpeto-encephalitis group, although they differed in some details. Levaditi in 1925 (L 178) expressed the opinion that this virus closely resembled rabies virus.

Doerr and Zdansky (D 140) repeated Koritschoner's experiments in general, using his virus, but they were not convinced of the diagnosis of the patients from which it was isolated, and did not consider that it belonged to the group of herpeto-encephalitis viruses.

Schweinberg in 1925 (S 135) stated that the histologic changes, both in the human brains from which the virus was obtained, and in the brains of the experimental animals, could be interpreted as rabies quite as well as encephalitis. He reviewed the history of the cases in detail, and showed that they pointed to rabies. It is pointed out by Schweinberg, and also by the other investigators, that this virus is pathogenic for dogs, whereas the herpeto-encephalitic virus is not; and that when injected corneally in a rabbit it does not produce a keratitis as does the herpeto-encephalitic virus. Immunity tests were then carried out which convinced Schewinberg that the Koritschoner virus was really a "street virus" stock. (Takagi in 1926 (T 6) stated that it was a form of virus fixe.)

Flexner in 1927 (F 66) stated that this virus is a strain of rabic virus.

Kobayashi in 1925 (K 87) reported the results of his experimental work in the Japanese epidemic of 1924. After numerous unsuccessful attempts to secure a virus by inoculating rabbits subdurally or in the cornea with specimens of brain, spinal fluid, etc., it was found that the brain emulsion of one patient, a typical case both clinically and histologically, was capable of producing symptoms of encephalitis in rabbits

at the third passage. A part of the brain of this patient was taken to the Kitasato Institute, where further studies were made.

It was found that the dog, cat, mouse, rat, guinea pig and pigeon were all susceptible to the virus after passage through rabbits. Corneal inoculation was not so successful as subdural and it did not produce herpes of the cornea. The author stated that the symptoms in the dog, cat, rat and mouse closely resembled those following inoculations with fixed rabic virus.

The virus was not filtrable through a Berkefeld N or Chamberland L_2 or L_3 filter; it was preserved in 33 or 50 percent glycerin for two months, but with some loss of virulence; it was not killed by heat at 55° C. for thirty minutes, but was destroyed at 60° C. for thirty minutes.

Cell inclusion bodies closely resembling Negri bodies, but smaller, were found in the brain of both the human case and the dogs and rabbits.

Cross immunity tests showed the same reaction with both rabic virus and this Japanese virus.

However, the author pointed out: (1) that in the rabbit the distribution of the rabic virus was mainly in the cord, while that of the "encephalitic" virus was mainly in the brain; (2) that the course of the disease with the rabic virus was much more acute than with the "encephalitic" virus; (3) that the rabic virus was more pathogenic for dogs than the "encephalitic" virus by subdural, and especially by corneal, inoculation.

A portion of this virus was sent to Dr. Flexner and was examined by Cowdry (C 236). After extensive experiments in cross immunity with this virus and a fixed rabic virus he concluded: "Since the symptoms produced in experimental

animals by the encephalitis virus of Kobayashi and by the virus of rabies are similar, and are accompanied by lesions which are indistinguishable, and since a cross immunity is demonstrable between the two viruses, the conclusion is advanced that the specimen of so-called encephalitis virus isolated by Kobayashi is in reality a specimen of rabic virus."

Group B.—Viruses which cannot be exactly classified.

Several groups of viruses have been isolated which have not been studied in such a way that it is possible to classify them with the herpes-like viruses to be described in the next section. The most important of these viruses are those isolated by McIntosh, both on account of the priority of the first virus isolated by him, and of the unquestioned reliability of his work.

In 1920 McIntosh and Turnbull (M 222) reported the inoculation of two monkeys, September 19, 1919, with the brain emulsion of a fatal case in the Derby epidemic. One monkey, a *Macacus rhesus,* was discarded, as it was found to have tuberculosis. The second, a *Cercopithecus patas,* inoculated with one cc. intracerebrally and five cc. intraperitoneally of filtered brain emulsion died November 14, 1919. Except for a "fit" on September 25, accompanied by fever, tremulousness and excitability, the animal was quite well until November 6. On that date a severe "fit" occurred, and after this there was rigidity, trembling, a tendency to lethargy, and disinclination for food. At necropsy nothing abnormal was found in any of the internal organs, and no gross lesions of the brain, except some congestion of the grey matter. On microscopic examination very definite lesions were found which are described in detail. These consisted, in brief, of perivascular infiltration and focal areas of infiltration located

mainly in the left basal ganglia and in the junction of the left basal ganglia with the mid-brain. These lesions were considered by McIntosh to reproduce the chief characteristics of human epidemic encephalitis.

An emulsion of the brain and cord of this *patas* monkey was injected intracerebrally and subcutaneously in three monkeys.

The conclusion of the experiments was reported by McIntosh (M 218). Two of these monkeys (*Macacus cynomolgus*)—the third monkey was not referred to in the second paper—were reinoculated fourteen weeks later. After the second inoculation one of the monkeys developed symptoms and was killed in about two months. Characteristic lesions were found in the brain. An emulsion of the brain and cord of this monkey was injected intracerebrally and subcutaneously in three rabbits. Two animals developed suggestive symptoms. One recovered, the other was killed on the eighth day after inoculation and showed histologic lesions in the brain. An emulsion of the brain and cord of this rabbit was injected intracerebrally in three other rabbits, one of which developed similar symptoms.

A third monkey (*Macacus cynomolgus*), which was kept as a control with an inoculated monkey, developed practically the same symptoms and died. The autopsy showed normal organs, except for a slight congestion of the brain. A microscopical examination of the brain revealed lesions in the basal ganglia. The facts would seem to justify the conclusion that this monkey contracted epidemic encephalitis from his association with infected animals. It is also interesting to note that he developed symptoms earlier than the inoculated monkeys, indicating that the monkeys were infectious some time before the development of symptoms.

Later, in 1923 and 1924, McIntosh (M 219) reported the successful inoculation of rabbits intracerebrally with brain emulsion in three out of five cases, and with spinal fluid in one out of three cases. The conclusions were based both on the symptoms developing in the rabbits and the histologic changes in the brain. Attention was called to the possibility of spontaneous encephalitis developing in rabbits, so it is certain that this factor was ruled out in these experiments.

Articles published by McIntosh, by himself in 1927 (M 220, M 223), and in collaboration with Scarff in 1928 (M 221), emphasized the care that had been taken in eliminating spontaneous encephalitis in rabbits as a possible source of error in the experimental work, discussed the possible relationship of the virus of epidemic encephalitis and herpes virus, and described the histology in detail.

Netter, Césari and Durand (N 73, N 74) in 1921 reported that in two cases out of ten they were successful in transmitting the disease to the rabbit by subdural inoculation with the filtrate of a brain emulsion. With the brain and salivary glands of the rabbit, other rabbits were inoculated successfully up to the fourth passage. The virus retained its virulence after being kept on ice for thirty-two days in 50 percent glycerin.

In Japan a number of viruses were isolated in the epidemic occurring in the summer of 1924. The exact nature of these viruses, except Kobayashi's, has not been determined and the problem is made more difficult by a recent article by Kaneko and Aoki (K 14a). These writers suggest the possibility that two kinds of encephalitis have been prevalent in Japan: one type, the same as that occurring in the western hemisphere, designated as Type A; the other, more or less endemic in Japan, but sometimes, as in 1924, reaching serious epidemic

proportions, designated as Type B. However, Flexner (F 66), after examining specimens from the brains of fatal cases of the epidemic of 1924, has stated that the lesions closely resemble those in epidemic encephalitis in Europe and America, and that the disease was probably identical with that occurring elsewhere.

Perhaps the most important work was done by Takagi (in some references spelled Takaki). In 1925 (T 5) he reported the isolation of six strains of virus from eight fatal cases by inoculation in rabbits. It is stated that 500 rabbits were used in the experiments, of which 58 percent gave positive results. The results were regarded as positive when (a) the inoculated animals had typical symptoms, (b) the brain did not show micro-organisms by culture, and (c) it was possible to transfer the virus to the next animal.

It was stated that an emulsion of the brain, proved sterile by culture, was used for inoculation into the brain, eye, conjunctiva, nose, testicle and skin. Positive results were obtained by all these methods, except the last. It was not stated whether all these methods were used successfully with the emulsion of the human brain, or only with the virus of passage.

The symptoms included sluggishness, emaciation, increased salivation, paralysis and fever. The animals died in from two to four weeks after inoculation. Experimentally, the virus was found in the brain, spinal fluid, blood, liver, spleen, kidney and salivary glands. Conjuctival inoculation produced no local lesions. When inoculation was made into the eye, an iritis developed, followed by glaucoma and atrophy. The symptoms, the lesions in the eye and the duration of the disease, are therefore quite different from those of the herpes-like encephalitis virus. Cross immunity tests also showed that this virus was different from that of herpes.

It was shown that the serum of convalescent patients contained immune bodies with respect to this virus. Immune bodies were also found in the blood and brain of inoculated animals.

The virus was filtrable, transferable in passage, was preserved in 33 to 50 percent glycerin for three months, was sterile by culture, and was killed at 55° C. in an hour.

In 1926 the main facts of the article just reviewed were reprinted in Vienna (T 6) followed by two other communications, the third being written in collaboration with Bonis and Koref (T 7).

The virus isolated by Takagi was named the virus of encephalitis japonica, as it was not entirely certain that the disease in Japan was identical with that occurring elsewhere.

The virus of encephalitis japonica and also the virus of herpes japonica were compared with various other viruses: Levaditi's encephalitis virus, Levaditi's herpes virus, the viruses isolated by Doerr and Lauda, Koritschoner's virus, Silberstein's virus and others.

After studying the symptoms produced in rabbits by the inoculation of these different viruses, and after making extensive tests in regard to immunity and virulence, the author concluded that the various herpetic-encephalitic viruses, with the exception of the virus encephalitis japonica, could not be differentiated experimentally nor serologically; that the virus of Silberstein did not agree with the herpetic-encephalitis viruses; that the virus of Koritschoner was related to virus fixè; and that the virus encephalitis japonica was easily differentiated from the other viruses and must be considered independently.

In view of this Takagi was of the opinion that the Japanese epidemic was a specific, independent disease.

Until more work is done with the virus of Takagi, the entire question must remain in doubt.

Nishibe (N 108, N 108a) in 1925 reported that fifteen strains of virus (ten from the brain, four from the spinal fluid, one from the saliva) had been isolated in Japan in 1924 from cases widely distributed. Apparently these included the six strains reported by Takagi.

These studies were mainly on the histo-pathology of experimental encephalitis in rabbits.

In order to test a theory propounded by Miyagawa, that certain organ and tissue cells give rise to destructive changes in the corresponding organs and tissues of injected animals, rabbits were inoculated subdurally and intraocularly with a brain emulsion of healthy rabbits. No lesions were found in the brains of these animals.

Nishibe stated that the strains were very similar. He called attention to the existence of spontaneous encephalitis in the rabbits of Japan. He stated that for this reason meningeal and perivascular infiltration could not be taken as criteria of the successful production of experimental encephalitis. He relied rather on the marked degeneration of the ganglion cells, especially those of the mid-brain and thalamus. He commented on the fact that the lesions produced by the encephalitis virus and the herpetic virus could not be differentiated microscopically. In 1926 (N 109) he emphasized these changes in the nerve cells as a differential point in distinguishing the lesions produced by the herpes and by the encephalitis viruses.

Flexner (F 66) was of the opinion that too much reliance should not be placed on these changes in the nerve cells, especially as such changes are not characteristic of the lesions in the human brain.

Komatsu and Yamasaki in 1925 (K 96) reported the suc-

cessful isolation of a virus or viruses (the number is not clearly stated) using the brain tissue, spinal fluid and spinal cord from five fatal cases. Rabbits were inoculated in various ways with this material, with the development of symptoms and histologic changes in the brain in about 10 percent of the animals. The symptoms were similar to those described by Takagi. The virus could be carried on in series. The incubation period was usually from seven to thirty-two days. Attempts to produce the disease in two Japanese monkeys were unsuccessful.

The investigators mentioned spontaneous encephalitis in rabbits and stated that they did not believe that this accounted for the histologic changes they observed.

Ito, Tsuchiya and Nakajo in 1925 (I 14, I 15) wrote that they were able to obtain viruses by subdural and corneal inoculations in rabbits with a filtered emulsion of the brain of three fatal cases of epidemic encephalitis. The animals that reacted died in from four to thirty days. The symptoms were similar to those described by Takagi. Histological changes were found in the brain.

The authors' conclusions were:

1. The disease is infective to the rabbit and transmissible in series.

2. The virus is filtrable.

3. The causative factor in the present epidemic has no relation to the herpes virus, as none of the rabbits developed herpes of the cornea.

In the second paper the virus was studied more in detail, as were also certain cultivable bacteria which had occasionally been isolated from human cases and rabbits. From these studies the following conclusions were drawn:

1. The virus is filtrable and can be transmitted in series.

2. The virus resists heat at 60° C. for twenty minutes, but is killed at 80° C. for twenty minutes.

3. The virus is infective for guinea pigs and rats.

4. It is not found in the blood, spleen or liver of the rabbits.

5. Immunity cannot be demonstrated in an inoculated rabbit in from thirty-nine to sixty-five days.

6. Eleven strains of bacteria, isolated from human cases and rabbits, were not considered as etiologic factors.

7. Filtered emulsion from the rabbit brain was culturally negative.

8. Encephalitozoon cuniculi were not found in smears or sections.

9. A form of inclusion bodies was found in the nerve cells at the angle for the hippocampus.

Kojima and Ono in 1925 (K 92a) reported the successful inoculation of rabbits by the intracerebral and orbital routes, using fresh spinal fluid. The virus could be carried on in series from the brains of the animals. They were also successful with the brain of a fatal case, using both filtered and unfiltered emulsions.

It is impossible to state whether or not the viruses just described were included in the list of fifteen mentioned by Nishibe.

Kaneko and Aoki (K 14a) in 1928 published a comprehensive study of encephalitis in Japan. In this article a number of references are cited to publications in Japanese journals not available to us. It is probable, however, that the most important work done by the Japanese has appeared in the publications to which we have had access.

It is stated that Chuin and his co-workers succeeded in transferring the disease (Type B) to animals; that Tsuda

obtained positive results in from 42 to 71 percent of cases, with brain emulsion, spinal fluid and blood, using rabbits and monkeys; that Watanabe and his co-workers made a successful transfer to monkeys, using blood serum; and that Yamaoka was successful in producing the disease in monkeys in nine out of ten cases and in rabbits in six out of ten cases, following subdural inoculations, and in monkeys in eight out of ten cases and in rabbits in four out of ten cases, following ocular inoculations. No definite data are available regarding these viruses.

Bessemans and Van Boeckel of Belgium in 1923 (B 191) reported that of sixty-one rabbits and thirty-two guinea pigs inoculated in various ways with spinal fluid or brain emulsion from four cases of encephalitis, seven rabbits and two guinea pigs inoculated subdurally or intracerebrally died at the end of the third or fourth week. It was possible to transmit the disease by an emulsion of the brain to the third or fourth generation.

Danila and Stroe in 1923 (D 23, D 24, D 25, D 26) claimed that they had isolated from cases of epidemic encephalitis strains of virus which were encephalitogeneous but not keratogeneous. These strains were apparently derived from the brain of a patient in one instance, and in the other from a vesicle on a patient supposed to be suffering from an atypical sporadic case of epidemic encephalitis. They stated later that on further passages the affinity of these strains sometimes changed.

Sicard, Paraf and Laplane in 1923 (S 208) reported that an emulsion of the brain of a patient dying four years after the onset of epidemic encephalitis, was injected in the brain of six rabbits and on the cornea of two rabbits. Four of the animals died in forty-eight hours, and two in thirteen and

fifteen days. Transfers from these rabbits were successful. Histologically the rabbits' brains showed typical lesions of encephalitis.

Szymanowsky and Zylberlast-Zand in 1923 (S 409) stated that of fifty-five rabbits inoculated corneally or subdurally with brain emulsion, spinal fluid or nasopharyngeal discharges, eighteen gave positive results. It is stated that the course of the disease in the rabbits was both acute and chronic. It is probable that these chronic forms may have been due to spontaneous encephalitis. The authors called attention to the existence of this condition.

Ottolenghi, Brotzu and D'Antona in 1923 and 1924 (O 47) reported that by injecting spinal fluid in rabbits and guinea pigs, using the corneal or intracerebral routes, a virus was obtained which they called "Siena." In one instance they were able to produce a slight keratitis by corneal inoculation. This keratogenous property did not continue through successive passages, but the encephalitogenous power persisted. The authors found that other diseases of the central nervous system produced the same effect. There was no cross immunity between the herpes virus and the virus "Siena."

Zannelli and Santangelo (Z 5) in 1921 reported a high percentage of positive results, with both guinea pigs and rabbits, using spinal fluid or blood serum for inoculation subdurally in the anterior chamber of the eye, intraperitoneally, on the chilled nasal mucosa, and in the nasal submucosa. The incubation period was forty-eight hours to thirty-one days. They stated that the virus could be passaged through several generations, that it was filtrable and bacteriologically negative.

In 1923 Santangelo and Zannelli (S 54) reported the successful inoculations of three out of five spinal fluids in rabbits and guinea pigs, subdurally and in the cornea. They stated

that following the inoculation in the cornea a keratitis developed and the animal died in about two months. With the transfer stock a milder keratitis developed and the animal lived. The successive transfers of the brain, subdurally, showed attenuation, rather than increase, of virulence.

Lalung-Bonnaire and Pons (L 40) reported in 1923 that in experimental work done at the Pasteur Institute of Saïgon they were successful in producing encephalitis in the rabbit by intracerebral inoculation with the brain emulsion of a fatal case. The virus could be transmitted in series, the duration of the disease decreasing from forty-six to six days in the course of transfer. Attempts to transfer keratitis in series were unsuccessful after the third passage.

Joltrain and Hutinel (J 50) related the results of an interesting experiment, the conclusions of which are somewhat indefinite on account of the combination of two diseases. The spinal fluid of a patient, who also had syphilis, was injected in the brain of a rabbit. The rabbit died in a few days with encephalitis. A second rabbit, injected in the brain with spinal fluid, died in thirteen days. An emulsion of the brain of the first rabbit was injected in two rabbits, one of which died suddenly. The other developed symptoms—a rise in temperature, somnolence and myoclonic spasms. It apparently recovered in about twenty-five days, but five months later lost weight and died with myoclonic spasms. An emulsion of its brain was injected in another rabbit, which died in thirteen days. This would weigh against spontaneous encephalitis as the cause of death in the preceding rabbit. A rabbit in the cage with the rabbit suffering the relapse was with young. Out of five young rabbits three died at once. A fourth was normal for two and a half months, when it suddenly developed trembling and myoclonic spasms and died in seven

days. An emulsion of its brain injected intracerebrally in a rabbit resulted in death one month later. The brain emulsion of this rabbit, injected in the brain and cornea of two other rabbits, resulted in death. The corneal inoculation was followed by keratitis. The authors concluded that the pregnant rabbit contracted encephalitis from her cage mate and transmitted it to her offspring.

The short period of incubation in some of the rabbits in this series and the production of keratitis would seem to show that the authors were not dealing with spontaneous encephalitis.

Boutroux in 1921 (B 333) reported the inoculation intracerebrally of a rabbit with the filtrate of washings of the nasopharynx and an emulsion of the brain substance of a patient, who also had a positive Wassermann reaction. The animal died in seven or eight days and the histologic lesions were considered typical of experimental encephalitis.

Dechaume (D 55) in 1927 reported a patient with epidemic encephalitis who gave a positive Wassermann reaction only in the spinal fluid. The histologic lesions were those of epidemic encephalitis, and the author concluded that the positive Wassermann reaction was only incidental. He quoted Bériel and Guillain, who agreed that the spinal fluid may show a positive Wassermann reaction in epidemic encephalitis.

Experiments were performed with the brain emulsion of this patient by Dechaume and Lebeuf (D 57). Inoculation was made in the menigeal space of young rabbits. One rabbit developed convulsions and torpor and died in seven days. The virus was passaged through several rabbits. The material was negative bacteriologically. It was not filtered.

Gerbasi and Guiffré in 1926 (G 52) reported the successful inoculation of rabbits with spinal fluid from four cases

and the brain substance of a fifth case. The inoculations were intracranial or corneal. Keratitis sometimes followed the corneal inoculations. In one instance the virus could be passaged. The authors stated that the brains of all animals were carefully examined and that there was no evidence of spontaneous encephalitis. Anaerobic cultures were made from the materials used in inoculation and positive results were obtained, with which the same results were obtained by inoculation as by the inoculation of the virus.

Blum in 1928 (B 247) reported the results of inoculation of material from the brain of a patient who had suffered from epidemic encephalitis for three years and who died of pulmonary tuberculosis. A virus was obtained by inoculating the scarified cornea of three rabbits. Keratitis occurred and the vesicles caused passages in two other rabbits. A fixed stock from the rabbit's brain was virulent after being kept in glycerin for one month. Unfortunately it was found to have lost its virulence after being kept in glycerin for two months, and it could not be reactivated. The lesions in the rabbits' brains were typical. The cultures were sterile.

From a study of these experiments it would seem that in several instances results were obtained that were sometimes definite and sometimes very suggestive of the isolation of a virus from patients suffering from encephalitis. Unfortunately the study of these viruses was not carried to a point that permits conclusions to be drawn in regard to their exact nature. These results do, however, add considerable weight to the theory that epidemic encephalitis is caused by a virus.

The extensive experimental work in connection with the Australian X-disease is referred to somewhat briefly, as the weight of opinion is that this disease was not epidemic encephalitis.

Flexner (F 64) pointed out that the histo-pathology of the lesions of the central nervous system in this disease resemble those of poliomyelitis much more closely than those of epidemic encephalitis, and that the seasonal occurrence corresponds with that of poliomyelitis. However, he called attention to the fact that the high mortality (70 percent), the small percentage of residual paralysis, and the occurrence of two known cases with signs of mental disorder following the disease, were unlike the usual manifestations of poliomyelitis. The age distribution was also somewhat different.

Moreover, in personal letters to the author, Dr. Cleland and Dr. Campbell have stated that they do not consider that the outbreak was one of epidemic encephalitis. They believe that it was a distinct disease more closely related to poliomyelitis than to epidemic encephalitis.

Breinl, in 1918 (B 371), reported the intracerebral inoculation in a monkey of 0.5 cc. of spinal fluid. The monkey became paralyzed and was chloroformed on the sixteenth day. Typical lesions were found in the brain. An emulsion of the brain and cord was injected into the brain of another monkey, which died of acute gastro-enteritis on the seventh day and showed no lesions in the brain.

An emulsion of the spinal cord of another fatal case was injected in a monkey. He became paralyzed on the twelfth day and was chloroformed five days later. There were diffuse infiltrations in the grey matter of the brain and cord and also perivascular infiltrations.

Cleland and Campbell (C 154, C 155, C 157) carried on very extensive experimental work in connection with this disease. With an emulsion of the brain and cord of three fatal cases, they were able, by intracerebral inoculations, to produce the disease in five monkeys (*Macacus rhesus*). (In the

monkey the disease ran a characteristic course, incoördination and violent, irregular muscular movements usually predominating over any paralysis or paresis.) Attempts to inoculate another specimen (*Macacus cynomolgus*) were unsuccessful.

The virus was conveyed by an intracerebral inoculation of the brain and cord from monkey to monkey through fourteen generations. The disease was transmitted by the same method from monkey to sheep ten times, from sheep to sheep, and back again to a monkey, from which a further series of monkeys was inoculated. The sheep showed a certain degree of immunity, about one out of three showing a definite "take." One horse and one calf were successfully infected with the monkey's brain.

The inoculation, with virus from the brain of a monkey, of four dogs, two kittens, three rabbits, two guinea pigs and one fowl was unsuccessful.

Intraperitoneal, intrasciatic and intravenous inoculations with virus-containing material, also intracerebral inoculations with spinal fluid, with a filtrate of faeces, and with nasopharyngeal swabs from human cases and contacts, all failed.

It was stated in 1919-20 (C 155) that the virus was held back completely or to a great degree by the Berkefeld filter, but, in a personal letter in 1928, Dr. Campbell stated that the virus was filtrable. Presumably later work was done which has not been published, or which at any rate has not come to our notice.

Storage of the virus-containing material in diluted glycerin in a cool place for more than a few days reduced or annulled its virulence. All bacteriologic work gave negative results.

Treatment of the virus-containing material with (a) normal human serum, (b) serum from recovered cases of poliomyelitis, and (c) serum from X-disease sheep, prolonged the

incubation period of the disease in the monkey, but did not destroy the virus.

It was found impossible to learn whether or not those who recovered from the disease later developed symptoms of the chronic stage, so characteristic of epidemic encephalitis. Dr. Cleland and Dr. Campbell made an effort to do so, but on account of the wide distribution of the patients in more or less isolated places, it was believed that it would be difficult. Dr. Cleland wrote that of the forty patients who recovered from the first outbreak, three had paralysis and two had signs of mental disorder at the time the report of the epidemic was written.

The Australian X-disease has been so widely discussed in connection with epidemic encephalitis that it has seemed best to include some data in this report. Many points—the clinical symptoms and after-effects, the high mortality, the symptoms of the inoculated monkeys, the failure of convalescent poliomyelitis serum to neutralize the virus, the production of the disease in sheep, a calf and a colt—seem to differentiate it quite definitely from poliomyelitis, in spite of the similarity in the histo-pathology described by Flexner. Competent observers, Cleland and Campbell, state that it was not epidemic encephalitis, and this opinion is borne out by Flexner's observations on its histo-pathology.

Was this disease then a distinct clinical entity, or is there a possibility that it was an unusual form of epidemic encephalitis? It has been the experience of all who have studied this disease that it shows wide variations at different periods.

If the Japanese outbreak of 1924 was, as is believed by many, a form of epidemic encephalitis, it certainly exhibited a marked variation from most other epidemics, both in its clinical features and in its seasonal occurrence.

The success that Cleland and Campbell enjoyed in isolating the virus, whatever the true nature of the disease, was doubtless due to the fact that the cases were so rapidly fatal. The successful inoculations were made with material from patients who had died in the second, third and fifth days, respectively, of the illness.

Group C.—Viruses closely allied to, or identical with, the herpes virus.

This group of viruses is of the greatest interest and importance in relation to the question of the etiology of epidemic encephalitis. They have all been carefully studied in many details and their close relationship to the virus of herpes has been established. Several of the viruses have been compared and have been found to be of the same general nature.

1. Levaditi and Harvier (L 184) were the first to isolate a virus belonging to this group. On February 10, 1920, an emulsion of the brain of a patient, who had died on the ninth day of the disease, was injected intracerebrally in two rabbits and a monkey. The patient had facial herpes, but the lesions in the brain were typical of epidemic encephalitis. The monkey and one rabbit showed no symptoms. The second rabbit died on the eighth day with symptoms and lesions of epidemic encephalitis. This virus was passaged in other rabbits and was named virus "C." A second virus, "Ch," of nasopharyngeal origin, was isolated a little later. This was obtained by rubbing the scarified cornea of a rabbit with a tampon of cotton which had been saturated with glycerinated water and left for twenty-four hours in the nostril of a patient. This produced kerato-conjunctivitis in the rabbit, from which transfers were made to the corneas of two other rabbits. Both these rabbits developed keratitis and one of them encephalitis,

with characteristic lesions in the brain. From the brain of this latter rabbit the virus was passaged and studies were made which proved its identity with virus "C." This virus was isolated in March, 1921, but it is best described in an article by Levaditi, Harvier and Nicolau in 1922 (L 197), and also by Levaditi in "Ectodermoses neurotropes" (L 174).

A third virus was isolated by Levaditi and Harvier (L 186) by inoculating rabbits intracerebrally with an emulsion of the brain of a patient with a choreic form of epidemic encephalitis. This was passaged for three generations, but it was evidently of a lower virulence than the viruses "Ch" and "C."

In 1921 Levaditi (L 169) stated that the isolation of a virus was made with great difficulty. After more than thirty inoculations in laboratory animals, using brain emulsion, spinal fluid, blood, saliva and nasopharyngeal secretions, only two fixed viruses were isolated. A third and less virulent virus was also obtained. This was evidently not so extensively studied. A similar virus was isolated in several instances from the saliva of healthy carriers (L 197).

Virus "C" has been most extensively studied by Levaditi and his co-workers.

Meantime in 1920, Doerr and Vochting (D 136), who were experimenting with the herpes virus, reported that certain of the rabbits with herpetic keratitis also developed symptoms pointing to a lesion of the central nervous system.

In 1921 Blanc (B 235) made the important observation that his experiments with the virus of herpes led him to believe that it was closely related to the encephalitis virus isolated by Levaditi. Blanc's virus was somewhat less virulent, but he suggested that the greater neurotropic power of Levaditi's strain may have been due to the more numerous passages in the brain of the rabbit.

Blanc's work was confirmed by Doerr and Schnabel (D 132, D 133, D 134), and by Levaditi, Harvier and Nicolau (L 174, L 195). It was shown that there was a cross-immunity between the virus of herpes and that of encephalitis and also that the histologic lesions in the brain were identical. The difference in the virulence of the two viruses and their cerebral and cutaneous affinities Levaditi (L 174) expressed by the following formula:

	Cutaneous affinity	*Cerebral affinity*
Virus of encephalitis.....	+	+ + + +
Virus of herpes.....	+ + + +	+ +

Numerous articles on the experiments, mainly with virus "C," were published in several medical journals. The earlier work is summarized in Levaditi's "Ectodermoses neurotropes" (L 174).

The more important conclusions in regard to its general nature are: The virus is filtrable; may be preserved for a long time in glycerin, milk and water; resists drying; is destroyed by phenol, potassium permanganate and bile and also by heat at 55° C.; it is active in a dilution of 1:1000 in saline; it does not diffuse readily.

Animals may be infected by inoculation in the brain, anterior chamber of the eye, peripheral nerve (the sciatic) on the scarified cornea, in the testicle, on the scarified skin, on the scarified or inflamed nasal mucosa or conjunctiva. If the virulence is high and a large quantity of virus is used, the disease may sometimes be produced by inoculation in the muscles of the neck or in the peritoneum. Intravenous, subcutaneous or intracutaneous inoculations are usually negative.

In experimental animals the virus was found in the brain and spinal cord. It was not found in the blood, spinal fluid, salivary glands, the internal organs, or in a filtrate of the nasal mucosa.

It was found that the virus was transmitted from the mother to the fetus, in the rabbit.

The results of attempting to neutralize the virus of encephalitis with convalescent human sera are summarized. The sera of six patients were used, and only one, that of a patient whose illness dated back more than a year, was found to have neutralizing power. The sera of patients more recently recovered seemed to facilitate the development of the disease in rabbits rather than to retard it.

Cross immunity tests with the virus of poliomyelitis and that of encephalitis proved the dissimilarity of the two viruses. Furthermore the virus of poliomyelitis does not produce a keratitis in the rabbit or monkey, nor can the disease be produced in the monkey by corneal inoculations.

The rabbit, the guinea pig, the monkey and the mouse were found susceptible to the virus. The white rat, the sheep and fowl were not susceptible.

The experiments with monkeys were summarized in 1924 (L 212). In 1920, a *Macacus cynomolgus* was successfully inoculated intracranially with virus "C." Typical lesions were found in the brain and the presence of the virus in the brain was proved by the successful inoculation of two rabbits. In 1923, three specimens of *Macacus cynomolgus* were inoculated cerebrally with virus "C." One died on the eleventh day, and characteristic lesions were found in the brain. Attempts to inoculate the brain in rabbits resulted in failure. Two chimpanzees inoculated with the same virus as the monkeys of the genus *Macacus* survived. In 1924, two specimens of the

genus *Cercocebus* were inoculated intracranially with "C." One developed the disease and died in nine days. The lesions in the brain were well marked and the brain was virulent for rabbits.

Commenting on the large percentage of negative results in attempting to inoculate monkeys with the virus, Levaditi stated in 1925 (L 178): "Therefore in general the monkey is refractory to encephalitis. This resembles the disease in man. Monkey and man are susceptible to encephalitis only if the natural refractory state is lacking. This explains the frequency of herpes and the rarity of encephalitis. The virus of herpes localizes easily on the skin, mucous membrane and cornea because these parts of the ectoderm offer in this regard a receptivity superior to that of the nerve axis. Modification of the 'soil' and increase in the neurotropic affinity of the germ are the essential factors which allow the disease to exist."

2. Doerr and Schnabel (D 134) in 1921 reported the isolation of a virus "Bâle I." Spinal fluid (0.2 cc.) from a case of epidemic encephalitis was injected subdurally in a rabbit. Symptoms appeared on the fifth day and the animal died on the following day. Transfers to other rabbits were readily made by injecting the brain emulsion of this rabbit subdurally. The incubation period varied from four to eight days and death occurred in from twelve to forty-eight hours.

The virus produced keratitis when used in inoculating the cornea of rabbits. It was pathogenic for guinea pigs and white mice. It was preserved in glycerin. Its presence was demonstrated in the blood of infected animals. Cross immunity experiments proved its identity with the virus of herpes.

3. Doerr and Berger in 1922 (D 131) related the isolation of a virus "Bâle II." The patient from whom the virus was obtained was a physician who died of epidemic encephalitis.

The symptoms and histologic lesions in the brain were typical. Attempts to inoculate rabbits, intracerebrally and corneally, with spinal fluid resulted in failure. An emulsion of the brain was prepared and nine rabbits were inoculated; three intracerebrally with fresh emulsion, three in the cornea with fresh emulsion, and three intracerebrally with the emulsion of a portion of the brain that had been kept for three weeks in diluted glycerin. Only one of the nine rabbits developed symptoms. This rabbit belonged to the first group. The symptoms developed eight days after inoculation, and death soon followed. The brain of this rabbit showed no organisms microscopically or culturally. It could be passaged in other rabbits by intracerebral and corneal inoculations. This last method resulted in keratitis. Cross immunity tests with a herpes virus proved the idenity of the two strains.

4. Berger (B 134) in 1922 reported the isolation of a virus "Bâle III." The histologic lesions in the brain of the patient from which this strain was obtained showed more involvement of the cortex than is usually the case in epidemic encephalitis. Spinal fluid was used to inject in seven rabbits, without success. The injection of an emulsion of the fresh brain was also unsuccessful. An emulsion of a portion of the brain which had been kept for fourteen days in glycerin produced encephalitis in rabbits after subdural inoculations. This virus could be transferred in series.

Zdansky in 1923 (Z 8) compared stocks "Bâle II" and "Bâle III" with each other and with a strain of herpes virus and found all three similar in their immunologic reactions, symptomatology and effects on the cornea. He also found that after intravenous injections of a dilute suspension of the brain in physiological salt solution, there was a localization in the brain in about 50 percent of cases, the incubation period

being prolonged. Levaditi had stated that intravenous inoculations were usually unsuccessful.

5. Schnabel in 1922 (S 101) described the isolation of the strain "Berlin." The spinal fluid from a patient in the acute stage of epidemic encephalitis was used to inoculate two rabbits corneally and four others subdurally. The spinal fluid was negative by cultural examination. One of the rabbits inoculated subdurally developed encephalitis on the seventh day and died on the following day. The brain was sterile by culture and perivascular infiltration was found in the midbrain and medulla. An emulsion of the brain produced keratitis when applied to the scarified cornea of rabbits. In one instance this progressed to a fatal encephalitis. The emulsion was also injected subdurally in rabbits with the production of fatal encephalitis. The virus was also pathogenic for guinea pigs and white mice. Cross immunity tests showed the close relationship between this virus "Berlin" and that of herpes.

In a later paper Schnabel (S 103) stated that the nuclear inclusions described in herpetic keratitis were also present in the corneal epithelium of rabbits inoculated with stock "Berlin."

6. Doerr and Zdansky in 1923 (D 137) reported that using an emulsion of a brain sent them by Kling, a virus "Hogander" was isolated by corneal inoculation of a rabbit. This virus resembled that of herpes, both in the symptoms produced and the reaction of immunity. In a later article (D 140) it was stated that the virus was not pathogenic for the dog.

7. Luger and Lauda in 1924 (L 347) inoculated a rabbit intravenously with the spinal fluid from a case of epidemic encephalitis. After an incubation period of eighteen days the animal developed symptoms. The virus "Wien" was carried on by subdural and corneal inoculations. The incubation pe-

riod diminished in the course of the transfers to four days. The virus was pathogenic for mice and guinea pigs.

This virus was apparently less keratogenous than the other strains described, but after a corneal stock was developed, it was easily carried on in series. Some of the animals with keratitis developed encephalitis with typical histologic lesions in the brain.

Cross immunity tests showed the close relationship between virus "Wien" and the herpes virus.

8. Perdrau in 1925 (P 83) reported the isolation of three viruses from the brain of three cases of epidemic encephalitis. A new technique was used in this work, which it seems desirable to give in the author's own words:

"Pieces of the mid-brain and of the basal ganglia from 3 acute cases of encephalitis lethargica were received preserved in glycerin. In none of these cases were any herpetic lesions noticed during life. Case 1 died 8 days, Case 2, 60 hours, and Case 3, 4 days after the onset of symptoms.

"Simple cerebral inoculations of emulsions of these brains into rabbits remained without result, when the material had been only 2 to 3 days in glycerin. Attempts were next made to increase the virulence of the virus either by adding to the inoculum the activating substance referred to already, or by the production of the latter in the animal experimented upon. In the first case the activating substance added to the emulsions of human brain consisted of an emulsion in normal saline of the brain of a rabbit which had received two sets of intradermal inoculations of the passage virus of herpes on 2 successive days, and which was killed 48 hours after the second inoculation. The brain of the rabbit was used after it had stayed at least 2 and not more than 3 weeks in glycerin in the ice-chest. The brain of an immunized animal acts

just as well, if not better, under similiar conditions of preservation, but there is a great saving of time by using the former. In the second case, that is to say for the production of the activating substance (henceforward referred to as an 'aggressin,' although its exact nature must remain in doubt for the time being) in the animal under experiment, emulsions of the human brain to be tested were injected repeatedly intradermally and intracerebrally, usually on alternate days, for 4 consecutive days, with or without the addition of the 'aggressin' in the case of cerebral inoculations.

"It should be stated that the inoculation of the 'aggressin' brain into rabbits by the various methods given here failed to show the presence of the virus itself in any of them, although an intradermal inoculation into a new rabbit usually leads to a little local redness. The same negative result attended the histological examinations.

"The results obtained with Case 1, being the most illustrative of the three, will be given in full, as considerable variations in virulence were noticed according to the length of time that the human brain had been kept in glycerin in the ice-chest.

"Case 1.—After 2 days in glycerin a single intracerebral inoculation remained without result. But when an intradermal inoculation was followed on the next day by an intracerebral inoculation, and on each of the next 7 days by cutaneous inoculations given by deep scarification, the scrapings of each scarification being used for the next, the rabbit developed the typical herpetic syndrome on the 12th day and died on the 14th day. The usual histological picture of a herpetic encephalitis was present in its brain and the virus was easily passaged. In order to determine whether the 'aggressin' which had been added to the inoculum for the cerebral inoculation

was responsible for the positive result, three rabbits were inoculated as follows with emulsions of the brain of the same case after it had been 17 days in glycerin: R 431 received a single cerebral inoculation of the human brain alone, R 432 received on 3 consecutive days an intradermal, a cerebral and an intradermal inoculation of human brain alone, respectively, and R 433 received a single intracerebral inoculation of a mixture of human brain and 'aggressin.' R 431 made a complete recovery and incidentally was found to have been immunised by the inoculation; R 432 and R 433 died in 6½ days with a typical herpetic syndrome. (Virus E.L. 1 is derived from R 432, in which no 'aggressin' was used.)

"This experiment clearly shows that the 'aggressin' cannot be the virus itself, especially in view of the fact that repeated inoculations of a concentrated emulsion intradermally and intracerebrally into new rabbits remain without effect.

"A point of great interest in the experiments just recorded is the reduction of the incubation period from 14 to 7 days, although the material had been longer in glycerin. When an emulsion prepared from the brain of the same case, after it had been in glycerin for 43 days, was injected intracerebrally into a new rabbit, the latter developed the typical herpetic syndrome and died within 4 days. It is clear, therefore, that there must have been a great increase in the virulence of the virus during the fifteen days that had elapsed since the last experiment. The emulsions used were prepared from small pieces taken from the same areas in every instance. The possibility of the virus having grown in the preserving fluid in the ice-chest is excluded by a large series of experiments in which liquid culture media containing glycerin were used. In every instance the virus was found to die rapidly in them and never to diffuse into the medium.

The most likely explanation seems to be that according to the theory of the production of an antibody in infected individuals the rate of degradation of the virus and of the immune substance, formed in response to the former, are not coincident. . . . .

"Case 2 died 60 hours after the onset of acute symptoms. Intracerebral inoculation of an emulsion of the brain of this case with the addition of some herpetic 'aggressin' as used for Case 1 led to no symptoms when the brain had been four days in glycerin. After 34 days in glycerin, intradermal and intracerebral inoculations given alternately on four consecutive days led to the production of the typical herpetic syndrome in 7 days. The histological examination showed an encephalitis of the usual type. Unfortunately owing to a shortage in the supply of animals the virus was not passaged immediately, and when it could be done the virus was found to be dead. At no time did a simple intracerebral inoculation of emulsions of the brain of this case produce any symptoms when used by itself. The amount of unbalanced virus must have been appreciably less than in Case 1.

"In Case 3, who died 4 days after the onset of acute symptoms, but who had probably been suffering from a mild infection for 5 months previously, an attempt was made to increase the virulence of the virus by intradermal passages. The first inoculation led to a very little local reaction, but the next two passages showed a very obvious one. At the third passage the presence of the virus was demonstrated by its ability to produce a very definite keratitis when injected by the corneal route. In this case the technique employed in Case 2 failed to give any results.

"It has therefore been possible by the adoption of a special technique to demonstrate the presence of a virus, indistinguish-

able from that of herpes, in the brain of all three cases of encephalitis lethargica in which this technique has been employed. From one of the cases (Case 1) a fixed virus has been obtained, which has been shown to be identical with that of herpes, including cross-immunity experiments. The only difference noted so far is a slightly greater virulence as shown by a shorter incubation period (4 days) in the case of the encephalitis virus (virus E.L. 1)."

Perdrau also stated that in the nasal washings of six cases out of seven, a virus was demonstrated capable of giving rise to a keratitis in the rabbit, and apparently identical with herpes.

The following table gives a list of nine viruses, closely resembling the virus of herpes, isolated from cases of epidemic encephalitis. The third virus isolated by Levaditi and the two additional viruses isolated from the brain by Perdrau might perhaps be included, but they were not so thoroughly studied.

HERPES-LIKE VIRUSES ISOLATED FROM MATERIAL FROM CASES OF EPIDEMIC ENCEPHALITIS

Investigator	Date	Name of virus	Material used
Levaditi and Harvier	1920	C	Brain
Levaditi and Harvier	1921	CH	Nasopharynx
Doerr and Schnabel	1921	Bâle I	Cerebro-spinal fluid
Doerr and Berger	1922	Bâle II	Brain
Berger	1922	Bâle III	Brain
Schnabel	1923	Berlin	Cerebro-spinal fluid
Doerr and Zdansky	1923	Hogander	Brain sent by Kling
Luger and Lauda	1924	Wien	Cerebro-spinal fluid
Perdrau	1925	E.L. 1	Brain

The number of viruses isolated and carefully studied is small in comparison with the numerous unsuccessful attempts

to transfer the disease to animals. A fairly large number of experiments that were suggestive, but not worked out in sufficient detail to be definite, were described under the heading Group B.

A theory to account for the large percentage of failures has been elaborated by Perdrau and by Levaditi. In 1922 (L 174) Levaditi observed that the virus of passage was more active after being kept for a time in glycerin and suggested that there seemed to be in the brain a substance tending to neutralize the virus, and that this substance was destroyed by glycerin. Perdrau in 1925 (P 82) stated that: "One may therefore conclude that the ultimate fate of the virus on reaching the central nervous system of patients suffering from encephalitis lethargica may be one of three:

"(1) A local cellular immunity may result, which is capable of overcoming the infection absolutely, with complete recovery of the patient.

"(2) A state of cellular immunity develops which can only keep in check, but not neutralize completely, the virus, and leads to a chronic infection, the virus being too attenuated to be capable of transmission to susceptible animals.

"(3) No immunity develops and the virus can be recovered from the central nervous system, the last being the least common of the three."

He elaborated this theory further in later articles (P 83, P 85).

Perdrau also believed that the antibody deteriorated in glycerin more quickly than did the virus, since, as he has already explained in the account of his experimental work, the virus in the human brain increased in strength after storage in glycerin. It will also be remembered that Berger had negative results after inoculation with an emulsion of fresh brain,

but after the brain was kept for fourteen days in glycerin the results were positive. Perdrau pointed out that if this theory is correct, the amount of virus in the brain will vary greatly. It is easy to see, therefore, how many negative results will be obtained, especially as the duration of epidemic encephalitis, before a fatal termination, is so variable. Perdrau also suggested that another reason for failure may be the difficulty encountered in infecting one species of animal with a strain of virus adapted to another species. This is true in passing from the rabbit to the guinea pig, for example, although these are species that are much more closely related than are the rabbit and man.

Levaditi, Sanchis-Bayarri and Schoen in 1928 (L 223a) developed a similar theory, "auto-sterilization of neuro-infections." They state that when the neurotropic ultra-viruses develop in contact with elements of the central nervous system, they react by lesions with a double significance—a nucleo-cytoplastic degeneration and a defensive process. This defensive process tends to the destruction of the virus in situ, a condition essential for cure. But when this process takes place in tissues so essential to life, the result may be the opposite because the intensity of the reaction may be incompatible with life. This would explain observations occasionally made—that animals die after inoculation with a virus (herpes, rabies, encephalitis) and yet the virus cannot be demonstrated in the brain by subsequent inoculations. The reaction has destroyed the virus as well as the inoculated animal. This process the authors call the "mortal auto-sterilization of neuro-infections." This would also explain the frequent failures to isolate the virus from persons dying with epidemic encephalitis, and the total failure up the present time to isolate the virus of post-vaccinal encephalitis.

Lépine in 1928 (L 145a) published an article tending to corroborate Levaditi's theory.

Two arguments are often advanced against the theory that the herpes-like virus is the cause of epidemic encephalitis. One is, that the patient from whom Levaditi isolated stock "C" also had herpes. This was not the case in the patients from whom the other strains were isolated. The other argument is that a herpes virus is sometimes isolated from the spinal fluid of patients not suffering from epidemic encephalitis. Flexner and Amoss (F 72) isolated such a virus from the spinal fluid of a patient who had cerebro-spinal syphilis. This virus "J.B." was carefully studied and found to be similar in all respects to the virus of herpes and that of encephalitis.

Ravault and Rabeau in 1921 (R 25) referred to a study made by them in 1903, which demonstrated changes in the spinal fluids of patients with genital herpes. Later experiments are also given in which the spinal fluids from five patients with herpes were used in inoculating the cornea of rabbits. Keratitis did not develop.

Bastai and Busacca (B 77) stated that they had demonstrated the herpes virus in the blood or spinal fluid of about 86 percent of twenty-one patients, many with herpes. Their results were not confirmed by other investigators. Schnabel (S 104), using the spinal fluid of forty-three patients with herpes; Doerr and Zdansky (D 140), using six brains, three from patients with herpes; Fischer (F 49), using serum or spinal fluid from forty-three patients, many of whom had a herpetic eruption or a history of herpes, all failed to isolate a herpes virus. Nicolau and Poincloux (N 100) were also unable to demonstrate the herpes virus in the spinal fluid of a patient with recurrent herpes.

Teissier, Gastinel and Reilly in 1926 (T 27a) were unable

to demonstrate the herpes virus in the blood of eight patients with herpes, or in the spinal fluid of fourteen such patients.

It would seem, therefore, that the weight of evidence is against the invasion of the central nervous system by the virus of herpes, except as a rare occurrence.

A certain amount of human experimentation has been done with Schnabel's strain "Berlin." Bastai and Busacca (B 78) inoculated with it the skin of three persons, who gave only a cutaneous reaction; the eye socket of a fourth, and the subarachnoid space of a fifth, without any reactions in the central nervous system. Schnabel (S 102) inoculated himself, with only a local skin reaction, using a virus of passage that produced fatal encephalitis in rabbits.

These negative results carry little weight against the etiological rôle of this virus in epidemic encephalitis, as many persons must have a high natural immunity to the disease.

The virus of passage of strain "C" has been injected intraspinally, for therapeutic purposes, in patients suffering from encephalitis. This injection is usually followed by a considerable reaction, and the vaccine is now given intramuscularly or subcutaneously. Nicolau and Poincloux (N 101) reported that spinal fluid, withdrawn as early as eight hours after the intraspinal inoculation of the virus, failed to give evidence by inoculation in rabbits of the presence of the virus. It is noted that in one patient a naso-labial herpetic eruption appeared after an intraspinal injection of the virus.

The identity or close similarity of the herpes virus and the various strains of virus isolated from patients with epidemic encephalitis has naturally led to extensive experimentation with the herpes virus.

While it is not within the scope of this monograph to study the details of the work on the herpes virus, a brief reference

will be made to a few of the more important publications. Levaditi has published very complete detailed accounts of herpes virus in "Ectodermoses neurotropes" (L 174) and "L'herpès et la zona" (L 180).

Blanc and Caminopetros in 1921 (B 236) demonstrated that the virus of herpes was filtrable; was sterile in culture; was infective for the rabbit, guinea pig and mouse, but not for the monkey (other workers have shown that the monkey may be infected), dog and pigeon; that the encephalitis produced in the rabbit by this virus resembled clinically and pathologically the encephalitis caused by Levaditi's virus of epidemic encephalitis; that a strong local reaction could give immunity against subdural inoculations. Also Blanc, Tsiminakis and Caminopetros (B 239) reported that the virus of herpes was rapidly destroyed by bile and that the serum of immunized rabbits and of patients convalescent from epidemic encephalitis did not neutralize the virus of herpes.

Perdrau in 1925 (P 82) carried out extensive studies on the immune reaction of the virus of herpes. He did not find the virus present in the spinal fluid of rabbits dying from encephalitis following cerebral inoculations. The virus was found to be more neurotropic when obtained from the brains of inoculated animals (the virus of passage), and more dermotropic when recovered from vesicles on the skin. A cerebral immunity was obtained by corneal inoculations with the aqueous humor; by intranasal inoculations of the virus of passage; and by cutaneous inoculations (either intradermal injections or rubbing the virus, either vesicular fluid or the virus of passage, on the scarified skin).

The cerebral immunity was best obtained by the intradermal injections of a neurotropic strain. One inoculation was

apparently sufficient. The active immunity appeared in about eleven days and lasted usually from one to four months.

Attempts to neutralize the passage virus with the serum of immunized animals failed, but the serum of one animal produced a longer period of incubation.

Attempts to neutralize the virus in vitro or to produce a passive immunity by emulsion of the brain of immunized animals were also unsuccessful. On the contrary, it was found that animals inoculated with mixtures of the immune brain and virulent brain died several days earlier than animals receiving the virulent brain only. This led Perdrau to formulate the theory that the brain of the immunized animal contained a substance that acted as an "aggressin." The action of this "aggressin" was more marked if the immune brain were kept for about two weeks in glycerin. Perdrau cited Levaditi's observations that a similar activating substance was apparently present in the serum of patients recently recovered from epidemic encephalitis.

The conclusion was reached that the cerebral immunity was of a cellular, rather than a humoral, nature.

A comprehensive study, "Contributions to the Pathology of Experimental Virus Encephalitis," was published in five sections, the first three by Flexner and Amoss in 1925 and the last two by Flexner in 1928 (F 72, F 73, F 74, F 69, F 70).

A detailed report was given of the isolation of the virus "J.B." from the spinal fluid of a patient with tertiary syphilis. This virus was found to be identical in all respects with the virus of herpes, and also with the viruses isolated from cases of epidemic encephalitis.

Numerous strains of herpes virus were studied and it was shown that they varied greatly in their virulence, and in their neurotropic and dermotropic properties. Two strains were

found that were more highly encephalitogenic than Levaditi's strain "C" and Doerr's strain "Bâle." This fact, the author believed, threw considerable doubt on the etiological rôle of this virus in epidemic encephalitis.

Flexner and Amoss reported that the serum of immunized animals possessed the power of neutralizing the virus. Cross immunity between the viruses of Levaditi and Doerr and the different strains of the herpes virus was also shown by the neutralizing power of the sera of animals immunized with the different viruses. Neutralization with human sera, either of normal individuals or of those recovered from epidemic encephalitis, was inconstant. This fact was considered evidence against epidemic encephalitis being caused by the virus in question. It was also stated that experimental encephalitis in rabbits, and epidemic encephalitis in man resembled each other only partially and in essentially superficial aspects.

In 1928 it was shown that strains of the herpes virus isolated at different times from the same patient varied greatly in their virulence and in their neurotropic and dermotropic properties.

In 1928 experiments were undertaken to test the theory advanced by Rose and Walthard (R 243) and by Dmitrieíf (D 128), that strains of the herpes virus and of the encephalitis virus are "suppressed" when injected in the brain of the guinea pig; that a similar occurrence takes place in human epidemic encephalitis, and that this accounts for the failures that have so often followed attempts to transmit the virus of epidemic encephalitis to animals.

Flexner showed that the results obtained by those investigators had been due to their use of weak strains of viruses. He summarized the results of his own experiments as follows:

"The guinea pig is subject to cerebral and corneal inoculation of the herpes virus.

"The effects of the inoculations vary with the strength or degree of virulence of the virus.

"Weak strains of the virus are implanted on the cerebrum with difficulty and strong strains with ease.

"Weak strains are quickly suppressed by the brain and strong strains may be passed indefinitely from brain to brain of the guinea pig. Strains of intermediate potency can be passed for a limited number of times only. . . .

"The guinea pig serves even more definitely than the rabbit to distinguish grades of virus according to strength or virulence. There is no difference of kind but only of degree of response to inoculation of herpes virus in the rabbit and the guinea pig.

"The etiology of epidemic encephalitis has not, therefore, been brought appreciably nearer solution by experiments with herpes virus carried out in guinea pigs."

McKinley and Holden in 1926 and 1927 (M 227, M 227a) published studies of experimental encephalitis. In 1924-25 McKinley had carried out some experiments with Le Fèvre in Brussels, using a herpes strain isolated by Le Fèvre, and the Levaditi strain. It was found that these viruses became attenuated when incubated with fresh serum, or when emulsions were prepared in salt solution and kept at ice box temperature. With these strains, thus attenuated, unsuccessful attempts were made to immunize rabbits by gradually increasing doses of the virus. Attempts to culture both of these viruses were also unsuccessful.

The experiments were continued in New York to establish more definitely the close biological relationship existing between these two viruses.

Rabbits were injected subcutaneously with gradually increasing doses of the viruses, attenuated as in the earlier experiments. The animals received ten injections over a period of three months. It was found that the rabbits were highly immunized, and that cross-immunity existed between the two strains. The serum of the immunized rabbits was virucidal for the two strains of virus.

The herpes virus was found to be markedly susceptible to ultra-violet rays as generated by the Alpine sunlamp. Exposure to the light of an emulsion in salt solution for five minutes destroyed the virus. These workers were unable to repeat Perdrau's experiment, that of producing cerebral immunity by a single intradermal injection of a neurotropic virus.

McKinley and Holden also demonstrated that the serum of immunized rabbits conferred passive immunity upon rabbits.

Attempts to immunize sheep against the herpes virus were only partially successful.

McKinley in 1928 (M 225a, M 225b, M 225c) reported that he was unable to produce serum with virucidal properties for the herpes virus by the subcutaneous injection of this virus in horses. Herpetic encephalitis was developed in guinea pigs by feeding them herpetic rabbit brain emulsion. He was unable to immunize rabbits by the subcutaneous injection of emulsified brain substance (containing herpetic virus) treated with chloroform. He concluded that the virus was destroyed by the treatment with chloroform.

Zinsser and Tang in 1926 (Z 15) published the results of comprehensive studies on the virus of herpes along the lines of immunology. Three strains of herpes virus were used and they were found to be extremely resistant to preservation in

neutral glycerol in the ice box. Experiments in which the virus was obtained by grinding the brain in sand, confirmed a theory of the authors that some of the virus is intracellular. This would account for the irregular results reported in attempts to neutralize the virus in vitro by immune serum, as the intracellular virus would be protected from the action of the serum. They were able to immunize rabbits by corneal, skin, and intratesticular inoculations. The serum of these animals had the power of neutralizing the herpes virus. It was also shown that the brain of immune animals contained a limited amount of protective substance, this in contradiction of Perdrau's report that such brain extracts increased the virulence of the virus. (Zinsser suggested that Perdrau's results may have been due to the rapid deterioration of the virus in the saline in which the emulsions were mixed.) Attempts at passive immunization with immune serum were negative.

Active immunity was produced only when some reaction to the living virus took place. Experiments with phenolized virus (after the Semple method in rabies) were negative, as the phenol destroyed the virus.

Attempts to transfer to animals a virus from material from human cases of encephalitis, using the method described by Perdrau, were unsuccessful.

When partially immunized rabbits were infected with the herpes virus, the course of the disease was sometimes modified so that it simulated the clinical picture of human encephalitis. The possibility was suggested by Zinsser that "human beings may, by repeated skin infections, attain a not inconsiderable partial immunity to herpes virus, which would explain the nature of the clinical course (as in our partial immunity rabbits) as well as the innocuousness of direct injections of herpetic virus into man, as reported by Bastai and Busacca,

and the finding of herpes virus in human beings not suffering from lethargic encephalitis."

In 1928, Zinsser (Z 14a) published a very complete study of epidemic encephalitis in which the herpes virus was extensively discussed. A possible analogy between the bacteriophage and filtrable viruses was suggested.

Experiments in the inoculation of human beings with the virus of herpes have been carried out to some extent.

Grüter (G 160a), who in 1913, by producing vesicles on the cornea of a rabbit by the inoculation of fluid from human vesicles, was the first to prove that herpes was caused by an infectious agent, has conducted such experiments. (His work was confirmed by Löwenstein [L 304].) In 1922 he reported (G 160b) that he had scarified the cornea of thirteen blind eyes and inoculated them with the herpes virus. The results were highly positive in four, weakly positive in four, and negative in five, instances. Control experiments with the cornea of the rabbit were all highly positive. Corneal reinfection in man was mild, indicating that the human cornea was only slightly immune after recovering from herpes, while the rabbit's cornea was highly immune. One of his colleagues submitted to an intravenous injection of the contents of several herpetic vesicles and suffered no general reaction.

Grüter emphasized the fact that the experimental transmission of the herpes virus to the human mucosa of the eye, nose and mouth is harmless and never results in general infection showing clinical symptoms, and that even intravenous injection of the virus is without systemic effects. He concluded that the great differences between the behavior of man and animal with respect to the herpes virus necessitated the greatest caution in applying animal data to man.

In 1924 Grüter (G 160c) reviewed his earlier work and stated that the weaker strains of herpes virus could be made more virulent by brain passages and that the more potent strains could be weakened by being kept for some months in glycerin in a refrigerator or for some hours in a saline emulsion at 36° C. He therefore concluded that the morphologically different strains of virus were etiologically similar. He expressed the opinion that the theory that virus is the cause of epidemic encephalitis could be solved only by a positive inoculation test on man.

Nicolau and Poincloux in 1923 (N 99) reported the cutaneous inoculation of human beings, using virus from the brain and cornea of the rabbit and also from vesicles in man. It was found that the corneal virus produced more positive results than did the cerebral virus, as the former was better adapted to the ectoderm. The virus from the vesicles in man gave positive results in individuals who had not reacted to the virus taken directly from rabbits, showing that the virus was modified by a passage through a human being.

Teissier, Gastinel and Reilly in 1923 (T 27) stated that they inoculated the herpes virus cutaneously on the arms of seven patients ill for from five to thirty-three days with epidemic encephalitis. All reacted to the infection. The inoculation was repeated, using the fluid from the human vesicles, and the reaction continued positive for four or five transfers. The authors therefore concluded, having regard to the identity of the herpes and encephalitis virus, that an infection of the central nervous system in man evidently does not confer immunity to reactions of the skin.

It may be pointed out, however, that those individuals might not be expected to have an immunity to the disease, since they were still suffering from it.

Bastai and Busacca in 1924 (B 75) reported the intracutaneous inoculations with a virulent brain emulsion (rabbit). This was followed by a mild local reaction without vesicles. Examination of the spinal fluid showed that in two out of three such cases the herpes virus had passed over into the spinal fluid.

Corneal vesicles were also developed by inoculation of the herpes virus (the ordinary herpes virus, or virus obtained from herpetic keratitis in rabbits, or from rabbit brain). In five cases with experimental keratitis, the presence of the herpes virus was demonstrated in the spinal fluid.

Two patients were injected intraspinally with a small amount of a weak brain emulsion (rabbit) diluted with the patients' own spinal fluid. Headache and pain along the spinal nerves followed, but disappeared in two or three days. Lumbar punctures were performed at intervals and it was found that the herpes virus remained in the spinal fluid for some months.

The authors therefore concluded that the herpetic infection not only became generalized, but might remain in the human organism in a latent state. They doubted the relationship of the herpes virus to epidemic encephalitis, believing rather that its occasional presence in the spinal fluid or central nervous system of encephalitis patients was purely accidental.

It would seem, therefore, that from the point of view of human experimentation there is no confirmatory evidence of the herpes virus being the etiological agent of epidemic encephalitis. This is not, however, a very potent argument against it, as many individuals are apparently immune to epidemic encephalitis.

The experimental work with monkeys affords more evidence of a relationship between the herpes virus and encephalitis.

Levaditi and Schoen in 1928 (L 225) published an account of an experiment performed in 1927. A specimen of *Macacus sinicus* was inoculated intracerebrally with an emulsion of rabbit brain containing the herpetic virus "N." The monkey developed symptoms on the ninth day and died two days later. The brain emulsion, used to inoculate rabbits intracerebrally, caused death from herpetic encephalitis. A histological examination of the monkey's brain showed characteristic lesions in the mesencephalon.

Zinsser has recently been carrying on experiments in producing encephalitis in monkeys with the herpes virus. His paper will appear in the *Journal of Experimental Medicine,* and it is not possible to publish the details at this time. However, through the courtesy of Dr. Zinsser and Dr. Flexner, we are permitted to use the following paragraph:

"Zinsser has been working with a species of monkey in which it is possible to produce both an acute and a subacute encephalitis by means of herpes virus. The details of these experiments are in press. On the basis of this work and his immunological experiments, he states that he is gradually moving in the direction of the view that since encephalitic disease is chiefly secondary to filtrable virus conditions, most of these agents may become neurotropic under circumstances of individual susceptibility, and that the difficulty of transferring to animals is very possibly due to a gradual loss of virulence consequent upon the prolonged nature of the cases which usually come to our hands at autopsy."

In spite of the vast amount of experimental work that has been done, there are still widely varying opinions in regard to the etiology of epidemic encephalitis. A comparatively small group believe that it is a toxic disturbance of the central nervous system. Another rather small group consider that a

pleomorphic streptococcus is the cause. A somewhat larger number of investigators are convinced that a filtrable virus, identical with or closely allied to the herpes virus, is the specific agent. In view of these conflicting opinions, many are forced to the conclusion that the etiologic agent of epidemic encephalitis has not yet been definitely demonstrated.

Chapter II

OTHER TYPES OF ENCEPHALITIS, INCLUDING POST-VACCINAL ENCEPHALITIS

The prevalence of epidemic encephalitis during the past ten or twelve years and the interest that has been aroused by this disease, with its protean clinical manifestations and its tendency to chronicity, has greatly stimulated the medical profession to the study of other forms of encephalitis. By far the most important of these is post-vaccinal encephalitis, which has been more or less prevalent for five or six years in England and on the Continent, especially in Holland. There are also fairly numerous references in the literature, especially recently, to encephalitis following measles, varicella, mumps, influenza and other acute infections. Whether more cases of this type have been occurring of late, or whether they are being better diagnosed, is a little difficult to decide. It seems certain, however, that there has been a very definite increase in the cases of post-vaccinal encephalitis in certain localities.

It is not within the scope of this monograph to discuss in any detail these other forms of encephalitis. Some of the recent references have been collected, however, and are included in the bibliography.

Rolleston in 1925 (R 225a, page 285) wrote that encephalitis followed measles more frequently than any other exanthema. Lust and Schick, discussing Lust's paper (L 353), each reported four such cases in 1926; Neal and Appelbaum (N 28), twelve in 1927; Musser and Hauser (M 425), eight in 1928; and Zinsser (Z 14a), four in 1928. Varicella is occasionally followed by encephalitis. Winnicott and Gibbs (W 119) described such a case in 1926 and reported other

cases from the literature. Winnicott (W 118) in 1928 reported a case of encephalitis following measles and varicella.

Zinsser (Z 14a) referred to cases of encephalitis following a variety of conditions: German measles, lead poisoning, pneumonia, bronchitis, acute gastro-enteritis and whooping cough.

Anderson in 1923 (A 102) and Brown and Symmers in 1925 (B 404a) reported an unusual type of acute serous encephalitis in children, characterized by a sudden onset, with sore throat, gastro-intestinal disturbance, convulsions, some indication of involvement of the central nervous system, high fever, and death in from twenty-four to forty-eight hours. The pathologic picture showed engorgement of the capillaries, perivascular and pericellular edema, cloudy swelling of the pyramidal cells and focal collection of glia cells, round cells, and a few leucocytes. Grinker and Stone (G 145a) in 1928 reported thirteen cases of acute toxic encephalitis in children. Their report contained both clinical and pathologic studies, and a review of the literature. The cases followed various acute infections; the upper respiratory tract, acute otitis media, mastoiditis, pneumonia, scarlet fever and septicemia. They pointed out that the pathologic changes in these cases of toxic encephalitis resembled those characterizing cases of acute lead encephalitis, as regards the vascular, glial and meningeal reactions. They quoted Hassin (H 90) to the effect that: "It seems permissible to claim that vascular infiltrative phenomena are indicative of an infectious type of encephalitis, while the proliferative phenomena denote a toxic condition." Grinker and Stone stated that the pathologic changes in none of these cases resembled those described by Brown and Symmers, and they quoted Symmers as stating that he had not encountered

any more such cases, and that he could not be sure that they constituted a new clinical entity.

In contrast with Grinker and Stone's report of the pathologic changes in their cases, one of which followed scarlet fever, are those found by Musser and Hauser, in two cases of encephalitis following measles, the latter more nearly resembling those of certain acute cases of epidemic encephalitis. There were many punctate hemorrhages and perivascular infiltrations with lymphocytes. A differentiation from other types of encephalitis was made by the authors: "There was one lesion, however, that was unlike any infectious encephalitis that we are acquainted with; namely, perivascular hemorrhages about some of the small vessels of the brain. In these there was apparently a diapedesis of erythrocytes, occupying in a circumferential manner the perivascular space. There was no diffusion of the erythrocytes beyond this space. A search for injury to the endothelial lining of such vessels did not show any lesion present. Apparently, the red blood cells escaped from the vessel because of the overdistention of the lining endothelium, or because of some other factor, probably toxic in character."

The etiology of these cases of encephalitis remains in doubt. Grinker and Stone believe that theirs were cases of true toxic encephalitis, the toxin produced by the micro-organisms which caused the initial focal lesion being carried to the brain, probably through the blood stream. Lust and Schick advance the opinion that the cause of post-measles encephalitis is a latent neurotropic virus which is stimulated to activity by the measles virus or by the lowered resistance of the patient.

Post-vaccinal encephalitis is by far the most important form of encephalitis, excluding of course the epidemic variety, both

as regards the number of cases and the bearing on public health, as it may affect the attitude toward vaccination.

Lucksch in 1924 (L 334) reported three fatal cases of encephalitis following vaccination, which occurred in Bohemia in 1923. These were reported in more detail by Lucksch in 1925 (L 335), together with some experimental work on the vaccine virus, which was found to be sterile and when injected in rabbits produced the usual reaction. Lucksch at first considered these to be cases of epidemic encephalitis, but later, in 1926 and 1927 (L 339, L 340), wrote articles in which he stated that these cases may be differentiated from epidemic encephalitis and poliomyelitis, both clinically and histologically. He therefore regarded this form of encephalitis as a distinct entity, and was inclined to be of the opinion that the vaccine virus was the etiologic agent. In his later experimental work, 1927, he was able to demonstrate the vaccine virus in the brains of three rabbits, after vaccination. This had already been done by other workers, Levaditi and Nicolau (L 214), Blanc and Caminopetros (B 237) and by Blanc, Melanidi and Stylianopoulo (B 238a).

Bastiaanse in 1925 (B 79) refered to a case reported earlier (in November, 1924) and discussed thirty-four other cases that had occurred in Holland from January 1, 1924, to June 30, 1925. In a later article in 1926, Bastiaanse, Bijl and Terburgh (B 79c) dealt with the subject more in detail. There were fifteen deaths among the thirty-five cases. All the patients were less than seven years of age, the majority being between three and five years. The symptoms developed in from nine to fifteen days after vaccination. The cases were distributed over twenty-four communities, the majority being in places of less than 16,000 inhabitants and in adjoining communities. Comparison with statisitcs of epidemic encepha-

litis for the same period showed that this disease was likewise more prevalent in smaller than in larger communities, and was epidemic in character. The histopathology of post-vaccinal encephalitis differed from that of epidemic encephalitis.

The vaccine virus used was prepared at three approved laboratories in different cities.

The authors experimented with vaccine virus used to inoculate rabbits in various ways, and after subdural inoculations were able to demonstrate a neuro-virus comparable to Levaditi's neuro-vaccine virus. They also demonstrated that vaccination did not immunize animals to the encephalitis virus. Experiments with the brain tissue and spinal fluids of fatal cases of post-vaccinal encephalitis failed to demonstrate the presence of any virus. Later experiments with the spinal fluid conducted by them with Levaditi, in Paris, failed to show immunity in animals to encephalitis virus or neuro-vaccine virus.

The authors reached the following conclusions:

1. No connection has been established between vaccination and post-vaccinal encephalitis;

2. The causative factor of post-vaccinal encephalitis is probably an ultra-virus;

3. It is probable that vaccination activates a latent virus present in the body;

4. Clinical, histological and epidemiological data, therefore, do not prove that the unknown factor is the virus of epidemic encephalitis, or that it is a related virus.

Terburgh in 1927 (T 28) reported 124 cases with thirty-eight deaths in the Netherlands, from June, 1923, to December, 1927. The highest incidence of the disease was in March. It was shown that immunity to post-vaccinal encephalitis is

greatest in the first year of life, and the vaccination of infants was therefore urged. He again stated his belief that the vaccine virus was not the primary etiological agent, but merely the cause of provoking a latent infection.

Bastiaanse in 1926 (B 79b) announced that the Royal Serological Institute at Utrecht had a serum against post-vaccinal encephalitis, which should be used immediately after the onset of symptoms. This serum is from animals actively immunized by cutaneous and intravenous inoculations.

Kraus and Takaki in 1925 (K 121) reported that they believe that they have proved, by complement fixation, that encephalitis following vaccination is not due to the vaccine virus.

Lucksch in 1927 (L 340), after making complement fixation tests, was not convinced that this method was without limitations in determining the forms of filterable viruses, but thought rather that it showed a close relationship between rabies, variola vaccine and herpes.

Turnbull and McIntosh in 1926 (T 115) made a clinical, pathological and experimental report of seven cases of encephalo-myelitis following vaccination. One case occurred in July, 1912, three in November, 1922, and three in September and October, 1923. In six instances death occurred during the acute attack of encephalo-myelitis, which was complicated by broncho-pneumonia. In one case death occurred later, when the lesions of the central nervous system were in a stage of healing.

The pathological changes were given in detail. They resembled the changes of non-purulent inflammation of the central nervous system, and in some respects were similar to those of epidemic encephalitis, except that the reaction was relatively slight in the basal ganglia and the most characteristic

histological feature was a zone of perivascular softening in the white matter.

The case examined in the subacute stage showed the same general distribution of lesions, but they were smaller and fewer, the foci of intense acute inflammation having commenced to scar, and the less intense, more diffuse infiltration having apparently undergone resolution.

Sections of the proximal and distal ends of the right and left brachial plexuses were made in three cases, to determine whether there was evidence of a spread of infection along the nerves to the central nervous system. No direct evidence of such an extension was found except in one instance, where the inflammatory reaction extended as far as the distal end of the left brachial plexus.

The authors discussed the close clinical resemblance of the cases and concluded that the vaccination was a definite causal factor; that the encephalitis might have been caused by the vaccinia virus, by some other virus introduced with it, or by the vaccination activating a virus latent within the patient, as suggested by Bastiaanse.

The authors compared the histological lesions with those of poliomyelitis and epidemic encephalitis, and concluded that post-vaccinal encephalitis could be definitely differentiated from these two diseases.

Extensive experimental work was done with material from several of the fatal cases. The presence of vaccinal virus in the left axillary glands of one case was demonstrated by Dr. Fildes by intratesticular and intradermal inoculations in a rabbit. Dr. McIntosh produced in two rabbits a mild meningo-encephalitis by injecting intracerebrally material from the spinal fluid of two cases. In one instance this could be passed on in series. Many other attempts were unsuccessful with

rabbits, and all were so with monkeys. Material from two brains, injected intradermally and intratesticularly, showed that the virus of vaccinia was present.

The authors called attention to the fact that cases, apparently similar, are reported of encephalo-myelitis complicating variola or varioloid.

Walthard (W 13a) reported experiments with three kinds of vaccine virus, including a neuro-lapine preparation of his own, similar to Levaditi's neuro-vaccine. These vaccines were used to inoculate rabbits in the cornea and guinea pigs in the skin, without producing encephalitis in the rabbits or myelitis in the guinea pigs. Two patients, vaccinated for the first time, failed to show vaccine virus in the spinal fluid twelve days after vaccination. The vaccine virus was found in the brain of a rabbit four days after corneal inoculation, but inoculation of this brain substance into other animals did not produce encephalitis.

Wilson and Ford (W 91a) in 1927 reported four cases of nervous complications following vaccination. Two cases, which recovered, showed sequellae. One had some mental defect, and marked obesity. The other had spastic paralysis. This is of interest because most writers have commented on the completeness of the recovery in recovered cases of post-vaccinal encephalitis.

Levaditi discussed the etiology of post-vaccinal encephalitis in 1926 (L 214), and again in more detail with Nicolau and Sanchis-Bayarri, in 1927 (L 215). Levaditi pointed out that he had never produced vaccinal encephalitis in rabbits by corneal or skin inoculations, either with ordinary vaccine or with neuro-vaccine. Walthard's results were confirmatory.

The occasional finding of vaccine virus in the brains of

fatal cases of post-vaccinal encephalitis or of animals after vaccination has little significance. Levaditi (L 214) had already shown that after experimental vaccination the virus is diffused throughout the whole body.

If the encephalitis is of vaccinal origin, then some stocks of vaccine should show neurotropic properties. Levaditi and Bijl did not find that this was the case with the stocks used in Holland.

Levaditi and his collaborators then proceeded to determine whether or not they could produce vaccinal encephalitis in simians by inoculating them in different ways with Levaditi's neuro-vaccine of passage. Three monkeys were inoculated intracerebrally with varying amounts of neuro-vaccine. They died in from five to eight days, and the presence of the virus in the brain, spleen and ovary was demonstrated by inoculation in rabbits. The lesions in the brains of these monkeys were strikingly different from the lesions of post-vaccinal encephalitis in man. They showed a hemorrhagic inflammatory meningitis of the pia mater, with a local vaccinal area. Another monkey was inoculated intravenously with the virus, to parallel the mode of infection in man. The monkey died in sixteen days with no trace of encephalitis.

This led Levaditi to believe that post-vaccinal encephalitis is produced by some other agent than the vaccine. A further proof of this is that after more than 20,000 vaccinations in Spain with Levaditi's neuro-vaccine, no case of post-vaccinal encephalitis developed.

Levaditi next considered whether post-vaccinal encephalitis might be due to an awakening by the vaccination of a latent encephalitic infection. He stated that:

(a) Clinically it resembles epidemic encephalitis very strikingly;

(b) From a pathologic standpoint there are striking resemblances;

(c) While inoculations in rabbits have all been negative as regards showing the presence of the encephalitis virus, the same is true with the majority of experiments with epidemic encephalitis;

(d) The morbidity of post-vaccinal encephalitis is like that of epidemic encephalitis;

(e) Vaccinal inoculations in the rabbit facilitate the localization in the nervous system of the herpetic encephalitis virus;

(f) The question may arise of the introduction of the encephalitis virus with the vaccine virus. Levaditi inoculated rabbits in the skin simultaneously with the stock C and the neuro-vaccine. Only a local reaction resulted.

Earlier, however, in 1925, Levaditi and Nicolau (L 213) had reported that the intracerebral inoculation of rabbits carrying the virus of the neuro-vaccine with human encephalitis virus gives rise to a vaccinal encephalitis by breaking down the natural resistance of the central nervous system, which opposes the entrance of the vaccine virus into the brain.

The authors, therefore, concluded that while the etiology of post-vaccinal encephalitis was not definitely proved, the condition was probably due to the arousing of a latent encephalitic infection, by the vaccination.

Levaditi, Lépine and Troisier (L 197a) in 1928 inoculated in various ways monkeys, rabbits, mice and chickens, with an emulsion of the brain of a fatal case of post vaccinal encephalitis. No trace of the vaccine virus or of any other virus was found. The animals were later vaccinated successfully, showing that no immunity to vaccine virus had been conferred.

Other animals were first vaccinated, and later inoculated, with the brain substance from the fatal case. There was no reaction except that two rabbits died, one from a contaminating infection; the other had vaccine virus in the brain.

Levaditi, therefore, concluded that the cause of post-vaccinal encephalitis is not the vaccine; nor is it, in the majority of cases, inoculable in animals.

Gildemeister and Herzberg (G 58a) in 1927 injected intracutaneously in one series of guinea pigs an emulsion of the brain of rabbits inoculated with Bâle III virus or with the contents of human herpes vesicles. A second series was immunized against foot and mouth disease. A third series was vaccinated against smallpox. The second and third series were later inoculated with Bâle III virus or with herpes virus. The second series gave the same reaction as the controls in the first series. In the third series the reaction was abortive in nine out of twelve animals, showing a certain relationship, as expressed by the immunity reaction, between the virus of vaccine and that of herpes or of encephalitis.

Zurukzoglu (Z 20a) reported several series of experiments concerning vaccine and herpes. In one series, four strains of vaccine were used, including two strains described as neurolapine and dermolapine. Rabbits were given massive doses cutaneously, subcutaneously, intratesticularly or corneally. In no case was an encephalitis produced. In the brain of one rabbit, inoculated in the testicle, the vaccine virus was found to be present. Complement fixation with one strain of vaccine was negative. In a second series it was found that when either vaccine virus alone was injected intravenously or herpes virus alone subcutaneously, the rabbits survived. When the two were combined, two rabbits out of three died with symptoms of herpetic encephalitis. Vaccine virus could not be

demonstrated in the brains of the rabbits, but a corneal inoculation with the brain emulsion produced encephalitis in another rabbit.

The authors, therefore, concluded that in animal experimentation vaccination may activate herpetic infections. Three series of experiments were performed on guinea pigs with the herpes virus, which have no particular bearing on this subject.

The last series concerned immunity relations between vaccine and herpes. The conclusions were as follows:

1. Rabbits' cornea, immunized to vaccine, showed weak partial immunity to herpes. The reverse experiment failed, because even minimal doses of the herpes strain used, provoked fatal encephalitis.

2. Guinea pigs immunized to herpes (plantar inoculations), showed distinct partial immunity to vaccine.

3. The reverse of this experiment showed a lesser immunity to herpes.

4. The sera of guinea pigs immunized to herpes were virucidal to herpes, and moderately virucidal to vaccine.

5. The sera of animals immunized to vaccine were considerably less virucidal to herpes than to vaccine.

Glanzmann (G 79) in 1927 published a review of the nervous complications of varicella, variola and vaccinia. He divided the cerebral complications of vaccinia into six forms:

1. Serous meningitis;

2. Encephalitis with hemiplegia or hemiparesis;

3. Encephalitis with trismus and tetanus-like manifestations;

4. Encephalitis with somnolence and cranial nerve palsies similar to epidemic encephalitis;

5. The encephalitis of Holland, characterized by vomiting, somnolence, convulsions, and high mortality with absence of paralysis and trismus;

6. Encephalo-myelitis disseminata, with meningeal manifestations.

He called attention to the regularity of the onset of symptoms, about ten days after vaccination, which indicated more than a chance connection with the vaccination.

He suggested the possibility that the cerebral complications occurring in these three conditions were due to an anaphylactic reaction in the central nervous system between the virus and the cell-adherent antibody. According to Sahli, an anaphylactic reaction quickly destroys the agent of the disease. This would account for the failure in most instances to demonstrate the vaccine virus in the brain of fatal cases of post-vaccinal encephalitis. From this point of view, the post-vaccinal nervous complications are comparable to the accidents of serum therapy.

The author stated that in his opinion more than a superficial similarity exists among these three diseases, varicella, variola and vaccinia.

Kraus (K 119) reported experiments with rabbits, guinea pigs and mice, using material from the brains of two fatal cases of post-vaccinal encephalitis, with negative results.

Mikulowski (M 301) in 1927 reported two cases of post-vaccinal encephalitis, both of which recovered, although one had residuals. He mentioned the possibility of a vaccinal allergy as the cause.

Wiersma (W 71) in 1927 called attention to the differences in the symptomatology of post-vaccinal encephalitis, as described by different writers. He also stated that the main differences, both in clinical and microscopical appearance, between epidemic encephalitis and encephalitis following vaccination, are shown by two facts: (1) the encephalitis after vaccination is a more acute infection, which is shown by its

usual rather stormy course, as well as by the presence of polymorphonuclear leucocytes in the perivascular infiltrations and by acute alterations in the ganglion cells; (2) this encephalitis is far more widely spread over the brain than is epidemic encephalitis, for symptoms occur which indicate lesions of the pyramidal tract, of the cortex, of the leptomeninges and of other regions, and in microscopical preparations alterations are found all over the white and gray matter.

The author considered Bastiaanse's theory of the vaccine virus stimulating to activity a latent virus, to be a reasonable explanation for these phenomena.

Pette (P 124) in 1928 published experimental studies on rabbits inoculated with vaccine and also with herpes virus. He found that a certain percentage of the animals inoculated with vaccine died in from four to fifteen days from a septic process due to the *B. bipolaris*. He showed that at a definite time following vaccination the *B. bipolaris* invaded the tissues of the animal from the nasal mucous membrane, where it had existed in the meantime as an avirulent germ.

In none of his animals inoculated with vaccine, corneally or intracutaneously, did encephalitis develop.

He therefore concluded that post-vaccinal encephalitis is a process engendered, not by the vaccine itself, but by some other agent.

Aldershoff and Pondman (A 78) reported experimental studies on post-vaccinal encephalitis. They injected in rabbits a Tarozzi broth culture of vaccine. One rabbit died seven weeks later from paralysis. Intracerebral injection of the brain emulsion of this rabbit in other rabbits produced death in one day. From the blood of these rabbits, *B. bipolaris* was cultivated. These findings suggested an inquiry into post-vaccinal encephalitis. The same organism had been found by

other workers without their attaching special significance to it. Pasteur had stated that this organism may produce a neurotoxin. This would explain why the brains of fatal cases have been found to be sterile. The authors found the same organism in rabbits that died after neurolapine vaccination. They referred to Pette's work, which showed that the vaccine virus may activate this organism in animals, and probably in human subjects also. The resulting toxins are then assumed to cause encephalitis.

The authors reported that rabbits die in from nine to fourteen days after subcutaneous neurolapine inoculations, but that if emulsions of the brains of these dead animals be injected into the brains of other rabbits, death follows in from one to two hours. This highly virulent agent the authors called "neurocidine," and they assume that it exerts a direct toxic effect, since a virus would hardly act so quickly. From their experimental studies, they are led to believe that there is a possible relationship among *B. bipolaris,* vaccine virus and "neurocidine."

Concerning a possible relationship between their experimental findings and post-vaccinal encephalitis, they state that Pondman reported an almost pure culture of the organism *B. bipolaris,* in the nasal mucus of a child with post-vaccinal encephalitis.

Euzière and Pagès in 1928 (E 74) reported six cases of nervous complication following vaccination. One patient had had attacks of labial herpes for two years. All the patients recovered. Four patients were treated intravenously or intramuscularly with arsylene-glucose. One of these patients was also given salicylated glucose intravenously. Another received luminal also, and a third uroformine.

The authors believed that encephalitis following vaccination was often purely coincidental, but that it might be due to an allergic action of the vaccine.

Bouman (B 313) in 1928 pointed out the difference in the histological pictures of post-vaccinal and of epidemic encephalitis. He expressed the opinion that the vaccine virus was not the direct cause of the post-vaccinal encephalitis, but that this was due to an unknown virus.

Nobel (N 117) in 1928 reported a case of post-vaccinal encephalitis, in a girl aged four, which developed ten days after vaccination. At the time of vaccination she had labial herpes. He concluded that, if the assumption is correct that there is an interrelation between herpes and encephalitis, a herpes rash contraindicates vaccination.

Mikulowski (M 302) in 1928 reported two cases of post-vaccinal encephalitis, in infants of thirteen and eight months of age. Both recovered, but the older infant had residuals. These cases are of interest because it is generally stated that post-vaccinal encephalitis occurs only in older children, and that, if recovery takes place, it is complete.

Gins (G 63) in 1928 stated that although ten million vaccinations have been performed in Prussia in the past fifteen years, only three cases of post-vaccinal encephalitis have been reported, although for the past ten years all illness following vaccination has been reportable. He called attention to the fact that cerebral disorders are not infrequent in unvaccinated children, and that they may follow other infections, such as grippe and measles. It is, therefore, quite possible that a child may be vaccinated, who already carries the germ of some other disease.

Winkler (W 116) in 1928 called attention to the German

public health law demanding the vaccination of infants within the first year of life, as compared with the Dutch law requiring vaccination before the age of six. Since post-vaccinal encephalitis has been reported far more frequently in countries permitting delayed vaccination, he urged compulsory vaccination in the first year of life, because: (1) infants still have a certain innate immunity; (2) their immature central nervous system is less susceptible to the virus causing encephalo-myelitis; and (3) they are less highly permeated than older children by all sorts of viruses, one of which may be activated by the vaccination, thus causing post-vaccinal disease.

Perdrau (P 86) published a study of the histology of post-vaccinal encephalitis. It was undertaken in order to corroborate the claims of Turnbull and McIntosh that post-vaccinal encephalitis can be distinguished histologically from epidemic encephalitis and from poliomyelitis.

The material from three fatal cases was studied in detail by special methods. Sections from nineteen cases of epidemic encephalitis were examined for comparison. The lesions were also compared with those of disseminated sclerosis and with the descriptions in the literature of those of nervous disorders complicating smallpox, measles, Schilder's disease and those occurring in anti-rabic treatment.

Perdrau concluded that post-vaccinal encephalitis is characterized by the presence around certain vessels of the central nervous system of an area of demyelination, described by McIntosh and Turnbull as extra-adventitial softening. Perdrau agreed with McIntosh and Turnbull that this lesion differentiates post-vaccinal encephalitis from epidemic encephalitis and probably from poliomyelitis also.

Perdrau expressed the opinion, from a study of the literature, that a similar lesion is found in nervous disorders fol-

lowing smallpox, measles and other fevers, as well as after anti-rabic treatments. This lesion is essentially similar to that of comparatively acute cases of disseminated sclerosis.

The suggestion is made that the agent responsible for the demyelination in all these various forms of nervous disorders may be identical.

Ledingham (L 91a) in 1928 called attention to the difference in the symptomatology of reported cases of post-vaccinal encephalitis, making about the same classification as that given by Glanzmann.

Olitsky and Long (O 21b) in 1928 were unable to produce encephalitis in the guinea pig by the combined action of Levaditi's strain "C" and a neuro-vaccine.

While this does not confirm the experimental work of Levaditi and Nicolau (L 213), it apparently does not controvert it, as there is so much difference in the reactions of rabbits and guinea pigs.

A report of the League of Nations Commission (111) on "Post-vaccinal encephalitis," called attention to several points in regard to this disease. Adults and infants seem practically immune, as nearly all cases occur between the ages of three and thirteen years. In most instances the vaccination had, to all appearances, run a normal course. Many different vaccine lymphs were used in the cases reported, and also different methods of vaccination. In some countries where primary vaccination is performed on children of school age, there has been little or no post-vaccinal encephalitis reported.

In conclusion the Commission points out:

(a) That cases of post-vaccinal encephalitis, as compared with the number of vaccinations, are rare;

(b) That the relation between vaccination and encephalitis is apparently not merely coincidental;

(c) That post-vaccinal encephalitis is a different disease from epidemic encephalitis;

(d) That the virus of vaccine, in the present state of knowledge, is not considered responsible for post-vaccinal encephalitis, but that an unknown factor exists, either a bacterial, a filter-passing or a latent virus, which by means of a reciprocal reaction causes the disturbance.

The Commission does not consider any of these conclusions as sufficient reason for discontinuing vaccination, which is the world's greatest weapon against smallpox. They urge primary vaccination during infancy.

Reported cases of post-vaccinal encephalitis:

Holland, 1923-27	139	cases
England, 1922-27	87	"
Portugal, 1922	1	"
Switzerland, 1924	2	"
Germany, 1924	3	"
Prague, 1924	8	"
Poland, 1927	2	"
United States, 1927	4	"
British Guiana, 1928	1	"

In 1928 the Ministry of Health of Great Britain published a comprehensive "Report on Vaccination" (114), by a Committee composed of Sir Humphry Rolleston, Dr. F. R. Blaxall, Dr. G. F. Buchan, Dr. A. E. Cope, Dr. Mervyn H. Gordon, Professor J. C. G. Ledingham, Dr. J. R. Perdrau and Dr. J. R. Hutchinson.

The Committee conferred with Surgeon-General Hugh S. Cumming of the United States Public Health Service, and Dr. Amyot, Deputy Minister of Health of Canada.

Part I of the "Report" deals with the subject of vaccination in general: the preparation, the testing and the standardization of vaccine lymph; practical methods of diminishing or removing any risks which may result from vaccination; methods of vaccination; and a brief reference to diseases of the central nervous system following vaccination.

Part II consists of the report of the Andrewes Committee, composed of Sir F. W. Andrewes, Dr. J. A. Arkwright, Dr. F. R. Blaxall, Dr. F. Griffith, Dr. J. C. G. Ledingham, Dr. J. McIntosh and Dr. E. Farquhar Buzzard. It deals entirely with diseases of the central nervous system following vaccination, reviewing both the literature which gives details of the cases reported in England, and also the experimental work.

An account is given of sixty-two cases, occurring from November, 1922, to February, 1924. While the majority of the cases occurred in children, there were seven cases from twenty to fifty years of age. The mortality was 58 percent. Ten of the recovered cases were followed up, and it was found that nine recovered completely. One developed hemiparesis, probably permanent in character. This is in contrast, of course, to the paralyses which often follow poliomyelitis, and the mental and other disturbances which often follow epidemic encephalitis.

As regards symptomatology, the Committee stated that: "In most instances the onset of symptoms was rapid, and the course of the disease acute. The predominant symptoms were of cerebral rather than of spinal origin, and included fever, headache, vomiting, strabismus and varying degrees of clouding of consciousness. Where paralysis of the limbs occurred, it was generally of the upper motor neurone type.

"In other words, these cases present the symptomatology of encephalitis, indicating a diffuse inflammation of the brain,

without special localization, and with very little evidence of involvement of the cord."

It is noted that in all instances the vaccination process was normal.

It is stated that of these sixty-two cases of post-vaccinal encephalitis fifty-three were vaccinated with lymph from the Government Lypmh Establishment. These cases were vaccinated with forty-five series of lymph. (A series is the product of one calf.) All these series belonged to a strain which had been in use since 1907, and with which about fifteen millions of people had been vaccinated. The source of the vaccine in eight of the remaining nine cases was given. In one instance it was unknown.

Post-mortem records were available in six of the fatal cases. Their histology was studied by Professor McIntosh and Dr. Turnbull, and also by the Committee. These reports have already been mentioned.

Professor McIntosh was able to demonstrate the presence of the vaccine virus in the brains of three fatal cases. No other virus was found.

Dr. Blaxall undertook experiments on animals, vaccinated in the usual way, to determine whether the vaccine was demonstratable in the tissues generally, and if so, for how long. Calves, rabbits and monkeys were used. The vaccine was found in various tissues, in the blood, the brain, the liver and the spleen, from the fifth to the thirteenth day, the times varying somewhat in different animals. This confirmed the observations of Levaditi and others as to the general dissemination of the vaccine after cutaneous vaccination.

The Committee then discussed three theories in regard to the etiology of post-vaccinal encephalitis. It is possible that: (A) The cases of encephalitis were genuine sequelae of vac-

cination, and due solely to the virus of vaccinia; (B) That the cases of encephalitis, though coincidental with vaccination, were due to some different and independent cause; (C) That the cases of encephalitis were due to the combined action of two morbid viruses, viz., vaccinia and some other familiar or unfamiliar virus.

After considerable discussion, the Andrewes Committee submitted the following report:

"(1) Among the 62 post-vaccinal cases of acute nervous disease, four were observed in hospital, while six were examined post-mortem and histologically, and the affection appeared to be fairly uniform, and, further, a total of 47 were, from the clinical point of view, possibly all of the same nature. In addition, another case similar clinically and histologically occurred in 1912. The scanty records, however, and the imperfect clinical accounts leave the question open as to whether even the majority were due to the same pathogenic causes.

"(2) The great increase in vaccination and the seasonal prevalence in certain areas of acute polio-encephalo-myelitis are facts which must be taken into account, though their effects cannot be definitely assessed.

"(3) It appears to be extremely improbable that the cases can have been due solely to the vaccine virus, and the majority of the Committee attaches no credence to this hypothesis.

"(4) It is not altogether improbable that vaccination may here and there have precipitated an encephalitis in a person harbouring another virus.

"(5) The rarity with which the factors necessary to produce such cases are simultaneously operative is shown by the absence of cases in other years, especially 1924, when their appearance was looked for.

"The Committee's reference was to advise as to research work which would be likely to elucidate the nature of post-vaccinal encephalitis.

"Ability to give such advice depended obviously on the conception formed by the Committee of the nature of these cases after full consideration of all the available data and of the circumstances under which they occurred.

"The Committee desire to report to the Minister that on the evidence submitted to and examined by them, or otherwise elicited—admittedly defective as that evidence may be in certain particulars—there would appear to have been in the cases concerned something more than the mere overlapping in time of vaccination with some familiar or unfamiliar neurotropic virus, and that the unusual clinical and histological manifestations and lethal character of the best studied cases suggest that a combination of vira of the kind indicated above was operating.

"The opinion thus expressed by them as a Committee is given without prejudice to any modification of this view that may be rendered necessary either by further observation and experience or by the outcome of experimental research applied to problems of this kind.

"*Suggestions for Future Research.*

"The Committee would suggest the more intensive study of neurotropic vira in general, with a view to improving the state of our knowledge concerning them, and, in particular, the close experimental study of any further cases of polio-encephalitis, which may occur in association with or independent of vaccination. To this end early information as to such occurrences is essential, together with full post-mortem examination by skilled observers, and immediate transmission to the laboratory of the material necessary for experiment. A

special problem, raised by the hypothesis put forward above, is eminently worthy of experimental investigation, namely, the effect of two vira acting concurrently. Thus, it should not be difficult to decide the question, whether or not vaccinia lowered the resistance of a susceptible animal to another virus, such as that of poliomyelitis or encephalitis lethargica, or even to ordinary bacterial infection."

This report was signed by all the members of the Committee except Dr. McIntosh, who submitted a minority report, which began as follows:

"Although I concur with the material embodied in the Majority Report, I am unable to subscribe to the conclusions expressed therein concerning the aetiology of the encephalitis. In particular is this true with regard to pp. 115, 121 and 122, in which the Committee has definitely set aside the hypothesis that the series of cases under investigation may have resulted solely from vaccination. Further, I consider that on epidemiological, clinical, histological and experimental grounds, the evidence is such as to exclude the possibility of either the virus of poliomyelitis or of encephalitis lethargica playing a part in the aetiology."

After some discussion he concluded: ". . . . three possibilities remain as to the aetiology of these cases:

"(a) That the vaccinia virus alone is responsible.

"(b) That the condition is due to herpes or to some unknown virus.

"(c) To a possible combination of (a) and (b).

"In the report, when discussing the hypothesis of cerebral vaccinia, the present-day state of our knowledge as regards the pathological effects of vaccinia or smallpox virus on the central nervous system was summarised, and it is unnecessary

to go further into the matter. It is certainly true that it is exceedingly difficult to produce brain lesions in series as Levaditi and Nicolau claimed to have produced. It is, however, possible by the cerebral inoculation of vaccine lymph to produce a very definite general meningo-encephalitis in animals, and in the case of the encephalitis under review, it is not a question of transmission in series, but of the occurrence of sporadic cases in relation to vaccination.

"Now, recent experimental work has shown that in ordinary vaccination there is a generalization of the vaccinia virus in all the tissues of the body, and from the nervous tissue of the cases of post-vaccinal encephalitis vaccinia virus was demonstrated and isolated. The somewhat indefinite encephalitis produced by the intracerebral inoculation of animals with the brain tissue from these cases is comparable with the results obtained when small doses of vaccinia virus are injected intracerebrally.

"It can be urged against this view that in ordinary circumstances vaccinia virus is present in the brain without producing any untoward effects, and this must be admitted. But it is also highly probable that new circumstances as to age, primary vaccination and extraneous circumstances (unknown virus, etc.) of which at present we are not cognisant, have played a part. On the whole the relative infrequency of these post-vaccinal cases of encephalitis suggests that vaccinia is only able to cause the condition in association with some accessory factor, such as indicated above. The postulation of an unknown virus does not help very much, however, toward a solution of the problem of the aetiology.

"The exact relationship of the virus of herpes is somewhat more difficult to define. We know from recent experimental researches that this virus on inoculation into rabbits can pro-

duce an encephalitis. According to the researches of Levaditi and Harvier, there is considerable reason for believing that this virus, if not identical with, shows very close relationship to, that of encephalitis lethargica. But, so far, the virus of herpes has not been isolated from the human brain or cerebrospinal fluid. Herpes virus, however, can produce a very definite keratitis, on scarification on the cornea of a rabbit. The material derived from cases of encephalitis under discussion, however, produced no such keratitis on inoculation, which is a fact somewhat against the view that herpes febrilis was the virus concerned.

"Therefore, before vaccinia virus can definitely be put out of account as a possible aetiological agent, more data will have to be obtained concerning the frequency of these cases and the neurotropic effects of vaccinia about which, at present, there is some difference of opinion."

In addition to the sixty-two cases already mentioned, twenty-five additional cases were cited, making a total of eighty-seven cases. Five of these cases occurred in 1926, and twenty during the first nine months of 1927. The age distribution of these cases was from nine months to twenty years. Twelve patients died and thirteen recovered. Of the latter, twelve recovered completely, and one, an adult male, had a very protracted convalescence and developed marked irritability of the urinary bladder.

Of of the cases reported by the Andrewes Committee, ten have already been mentioned as having been followed up, and subsequent histories were obtained of twelve more. Seven are described as completely recovered; in three, a slight mental change is reported; one had a repetition of symptoms twelve months after the first attack; and one had been subject to fits since his illness.

It would therefore seem that the earlier assumption of complete recovery following post-vaccinal encephalitis is not borne out by facts, since in this report of twenty-two cases which were followed up, there were after-effects in six.

The Report continues with references to cases reported in other countries. It is stated that the first recorded case of encephalitis following vaccination appears to have been reported by Comby in France, in 1905 (C 191a).

After some discussion the Committee concluded:

"Although further evidence has accumulated since the Andrewes Committee reported, no new facts have emerged, whether from epidemiological, clinical or experimental research, which enable us to weigh with greater precision the various hypotheses of causation which both the Andrewes Committee and Continental writers have discussed.

"We agree that on present evidence the association of encephalitis with vaccination is not fortuitous and that vaccinia cannot be held solely responsible for the nervous sequelae. On the other hand, when we come to weigh the hypothesis of combined virus action which appealed most strongly to the Andrewes Committee (at least as a provisional working hypothesis), it becomes extremely difficult owing to lack of essential facts to evaluate the hypothesis any more precisely than they did. They simply stated that the facts might probably find explanation on the assumption that vaccinia had the power to activate known or unknown neurotropic viruses that may be harboured by the vaccinee, and they therefore recommended the prosecution of experimental research directed towards the testing of this hypothesis. As is shown below, some preliminary experimental work on this point has been recorded, but it does not so far enable us to speak with authority on the possibility of combined virus action. The Dutch observers,

we understand, are inclined to dismiss encephalitis lethargica as playing a possible rôle in conjunction with vaccinia and they suggest that both in Holland and in this country some unknown and otherwise innocuous virus may be harboured by individuals, particularly in country districts, which has the property of being activated by vaccinia and possibly also by the viruses of measles and of whooping cough; by itself they conceive it to be a harmless saprophyte. Like the Andrewes Committee, the Dutch observers exonerate vaccinia as the sole cause of the condition. In the present uncertainty with regard to the etiology of encephalitis lethargica and of the cerebral manifestations of poliomyelitis virus it would, we think, be inadvisable on the available evidence to exclude these conditions from playing a possible rôle in conjunction with vaccinia, particularly as we are ignorant of the alterations in the clinical and histological pictures that combined viruses may produce. Only further experimental research coupled with the prompt investigation of any further cases that may occur will throw light on this problem."

The experimental work carried out by various investigators was reported, and also the work done by Dr. Blaxall and Dr. McIntosh, which has already been recorded.

The brains and cords of five additional cases were also examined by Dr. Blaxall, and the presence of vaccine virus was demonstrated in two instances. These same cases, however, yielded negative results when examined by Dr. Maitland at the Lister Institute.

A large number of experiments carried out with material from fatal cases, by Dr. Perdrau and by Dr. Maitland (on behalf of Professor Ledingham), to determine the presence of a virus, yielded negative or inconclusive results. Rabbits were used principally, also a few guinea pigs and one monkey.

Dr. Maitland and Dr. Gordon, the latter using a somewhat different technique, were unable to confirm Zurukzoglu's findings in regard to the virus of vaccinia increasing the activity of the herpes vrius.

Dr. Perdrau carried out experiments to determine the possibility of a mutual interaction of the viruses of vaccinia and herpes, or of vaccinia and the herpeto-encephalitis virus.

Four experiments are described, the results of which may be briefly summarized as follows:

1. The vaccinia virus was recovered from the brain of a rabbit seven months after intracerebral inoculation.

2. Can herpes virus and neuro-vaccine exist side by side in the brain of the rabbit? The results of the experiment suggested that the two viruses might co-exist for at least two passages, and that they can be separated by the use of animals immune to one or the other of them.

3. Experiments on cross-immunity between herpes and vaccinia. It was not demonstrated that such a relationship exists.

4. Activation of one virus by another. This was not demonstrated.

Dr. Gordon carried out a series of experiments on vaccine virus in relation to encephalitis. The results of this work were not sufficiently conclusive to necessitate discussion.

The pathology of post-vaccinal encephalitis was described by Perdrau. This has already been noted.

In considering the subject of post-vaccinal encephalitis, certain points seem to have developed as additional cases have been studied.

1. The age distribution is far wider than was at first stated. Cases have been reported in the first year of life, and also in older children and adults. It is obvious that the greatest num-

ber of cases occur in the years when vaccination is most likely to be made.

2. Although the first cases were originally diagnosed as epidemic encephalitis, it was said a little later that post-vaccinal encephalitis could be differentiated clinically from epidemic encephalitis. A study of more cases has shown that the symptomatology of post-vaccinal encephalitis varies widely. The protean clinical manifestations of epidemic encephalitis are well known. It would seem that a purely clinical distinction between these two diseases would often be difficult, if not impossible.

3. It was claimed at first that patients recovering from post-vaccinal encephalitis had no after-effects. The study of a larger group of cases revealed residuals in a number of instances.

These points are not emphasized with the idea of indicating that post-vaccinal encephalitis and epidemic encephalitis are identical, as it has been stated by excellent authorities that they can be differentiated histologically. It is merely desired to call attention to the complexities surrounding the study of the subject of encephalitis.

Chapter III

SUMMARY OF THE TREATMENT OF EPIDEMIC ENCEPHALITIS

The abstracts of the articles on epidemic encephalitis were prepared with the purpose of collecting as much definite information as possible with regard to the results of the very numerous methods used in treating the disease. A tabulation was made from the abstracts, collecting for each therapeutic agent the author and the reference reporting it; the dosage and method of administration, where these were given; the number of cases treated and the stage, when stated; and the results of the treatment or the comments of the author. This tabulation was then rearranged and recorded in the form in which it appears in this report.

Certain general considerations must be taken into account in connection with the subject of treatment. Many authors did not report definitely on the number of cases treated, and often the results were expressed in general terms. In practically no accounts were there any records of controls. The few containing such records were in connection with such drugs as hyoscine, atropine, stramonium, bulbocapnine, etc., for which only a symptomatic therapeutic value is claimed. It is probable that more weight should be given to the number of unfavorable accounts than to the number that are favorable, as physicains are much more likely to record successes than failures. Indeed many prominent neurologists have written very little on the subject of the treatment of this disease, and it is to be inferred that it is because no line of therapy has been sufficiently favorable to warrant a recommendation.

In summarizing the favorable and unfavorable results of or

comments upon, many therapeutic measures, it is a striking fact that very often approximately two-thirds have been favorable, and one-third unfavorable. These results have a remarkable parallelism with the mortality of the disease in the acute stage. While the mortality has varied greatly in different epidemics, a fair average is from 25 to 30 percent.

Lumbar punture has not been recorded, as it is obviously only a symptomatic measure. While a few reported unfavorably upon its use, the majority recommended it, if the pressure of the cerebrospinal fluid was increased.

Certain physicians have strongly advocated special forms of treatment, both in the acute and chronic stages. To what extent the personal equation may have entered into their interpretation of results, it is at present impossible to judge. This can be done only by administering these various measures to a group of cases, with an adequate series of controls.

In regard to the present status of the therapy of the disease, one may quote Hall (H 29a):

"The evidence as to the real value of any of the many methods of treatment advocated is difficult to estimate in a disease the natural progress of which is so extremely irregular. The verdict on the whole is that, at present, any reliable therapeusis, either for the disease itself or for its many after-results, does not exist."

Acriflavine, Trypaflavine, Septacrol*

The dye was usually given intravenously, in doses of from 5 to 25 cc. of a 0.5 percent solution, repeated six or more times.

No series of cases with controls is reported. While the comments on, or results in, the acute stage are, with one

* A closely related chemical containing silver.

Number of references	Number of cases	Stage			Comments of results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
30	115	45	65	5	Early	17	1
					Late	8	3
					Not stated	2	3

exception, favorable, the number of cases treated is too limited to make the observations of value. In the late stage there is even less evidence of value, as three out of eleven comments or results were unfavorable.

Summary Comment.—Up to the present time acriflavine and allied substances have little to recommend them in the treatment of epidemic encephalitis.

Adrenalin

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
71	80	24	11	45	Early	6	2
					Late	6	2
					Not stated	18	5

The drug was given by mouth, subcutaneously, intravenously, or intraspinally. It was sometimes used in combination with other agents, as strychnine, urotropin, sodium cacodylate, pilocarpine, jaborandi, morphine, or thyroid preparations. It was also used in physiological salt solution in hypodermoclysis, and as "adrenenalized glycerated serum."

Summary Comment.—There is no evidence to show that adrenalin is of any real benefit here, except possibly under certain conditions, as a symptomatic measure.

AFRICAN RECURRENT FEVER*

Number of references	Number of cases	Stage, late	Comments or results		
			Stage	Favorable	Unfavorable or doubtful
4	71	71	Late	3	1

* Caused by Treponema duttoni.

The three accounts, in which improvement is recorded in about two-thirds of the cases treated, do not indicate definitely the degree of improvement, its character, nor its duration, beyond two or three months. The fourth investigator reported unfavorable results.

The organism was kept alive in white mice, and 0.5 cc. of the blood of the mouse diluted to 10 cc. with normal saline solution was injected intramuscularly in doses of 1-2 cc. The recurrent fever is mild and spontaneous recovery follows. One report claims that this treatment has certain advantages over malaria.

Summary comment.—The evidence concerning this means of treatment is too limited at the present time to permit of definite conclusions. Such results as have been reported warrant its further use under carefully controlled conditions, and in a series of cases sufficiently extensive to make deductions therefrom of real value.

For those interested in this treatment, the following refer-

ences are given to the bibliography: M 73, B 215, H 183a, P 39a.

AMMONIUM BENZOATE

Number of references	Number of cases	Comments or results		
		Stage	Favorable	Unfavorable or doubtful
3	Not stated	Not stated	3	

Ammonium benzoate was used in combination with urotropine and sodium salicylate. All the references are given by the same physician, and favorable results are reported, but as the three drugs were combined, it is impossible to evaluate the efficiency of each one. Evidence is otherwise entirely unsatisfactory, as the number of cases is not stated, and no definite information is given.

Summary comment.—Reports on this drug are insufficient to recommend it.

ANTIMONY*

Number of references	Number of cases	Comments or results		
		Stage	Favorable	Unfavorable or doubtful
1	3	Late	1	

* Stibenyl.

The drug was used intravenously, in doses of from 5 to 30 cg. in three cases of Parkinsonism, with marked improvement

reported. The small number of cases makes the evidence insufficient and leaves the value of the treatment doubtful.

ANTIPYRINE

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
5	10	2	8	Early	.....	2
				Not stated	2	1

The drug was given intravenously once to a patient who died; otherwise it was presumably given by mouth. In one series in which six patients were treated, the antipyrine was combined with quinine and anti-pneumococcus serum. Another patient was treated by antipyrine and a combination of many other agents. No conclusions are justified concerning the value of antipyrine in the treatment of the disease process, though it may be helpful symptomatically.

ARNICA (TINCTURE OF)

There was only one reference to this drug, and it was apparently used in only one case. The effect was not stated. The conclusions to be drawn are obvious.

ARSENIC*

A great variety of preparations of arsenic were used. In certain instances it was simply stated that arsenic or arsenic compounds were employed. Sodium cacodylate was most commonly referred to, being mentioned seventy-one times. It was usually given hypodermatically in large doses, up to 1 gr.

* Salvarsan preparations are listed elsewhere.

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
146	142 plus several hundred	15	97 plus several hundred	30	Early Late Not stated	8 50 15	7 28 16

daily, but the dosage and the time interval varied greatly. It was occasionally given intravenously. In the large series of several hundred cases it was given intravenously in a 50 percent solution, 0.5 to 2.5 gr., three times a week. Arsenous acid was sometimes used, also hectine, galyl, Fowler's solution, iron cacodylate, a compound of arsenic and antimony, a combination of arsenic and phosphorus, and sodium cacodylate with scopolamine hydrobromate.

In the later writings there are references to arsylene (sodium cacodylate with an allyl radical substituted for an alcohol radical), which is given by mouth or hypodermatically, and, glucosated, intravenously, the dosage being 10 cc. of a 4 percent solution.

In most instances not enough cases were treated by any one group of workers to make the evidence very reliable, especially as there were no controls. In general the drug would seem to be useful in the chronic stage, but against this apparent evidence is the statement made in one reference that sodium cacodylate was used intravenously in "several hundred" cases in the chronic stage, and that the ultimate result was striking only in a few patients, particularly those with a positive Wassermann reaction.

Summary comment.—The weight of evidence in favor of the arsenicals is slight, the number of investigators favoring these being counterbalanced by an almost equal number reporting unfavorably. Arsenic evidently has no specific value, and the favorable reports may well be due to its general tonic effect.

ATROPINE

Number of references	Number of cases	Stage		Comments or results		
		Early	Late	Stage	Favorable	Unfavorable or doubtful
75	105	1	104	Early	1	
				Late	52	13
				Not stated	1	

The drug was given in the form of atropine, tincture of belladonna, or bellafolin. It was sometimes combined with morphine or hyoscyamus, bromide and chloral, and in one instance it was recommended to be given alternating with scopolamine. The atropine was given orally, subcutaneously or intravenously; the tincture of belladonna orally in doses of from 10 to 30 minims three times a day; the bellafolin, orally or subcutaneously.

Summary comment.—The reports on the use of atropine or its derivatives indicate that this drug has value in the treatment of symptoms in the chronic stage. Even here it is not unanimously endorsed. Hall (H 31) reports favorable results in Parkinsonism with large doses of the tincture of belladonna, from 10 to 30 minims three times daily.

AUTO-SERUM THERAPY
Blood Serum

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
36	156	32	114	10	Early	5	6
					Late	10	5
					Not stated	3	2

The treatment consisted in injecting the patient's own serum, either intraspinally in doses of from 10 to 15 cc., or in some cases intramuscularly.

Summary comment.—The evidence of the cases reported offers nothing to recommend this mode of treatment.

AUTO-SERUM THERAPY
Cerebro-Spinal Fluid

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
24	33	13	15	5	Early	5	4
					Late	3	2
					Not stated	3	3

The treatment consisted in most cases in intravenous injections, also intramuscular or subcutaneous injections, employ-

ing the patient's own cerebro-spinal fluid or that from convalescent cases, in amounts usually from 5 to 25 cc.

Summary comment.—The evidence of reported cases offers nothing to warrant the use of this treatment.

BISMUTH

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
4	2	2	Early	1	
			Late	1	1
			Not stated	1	

Bismuth was used twice, and a bismuth compound with iodine and arsenic once. The dose was not stated, nor the method of administration, except by one author, who injected it.

BLOOD TRANSFUSION OR HUMAN SERUM

Number of references	Number of cases	Stage	Comments or results		
		Early	Stage	Favorable	Unfavorable or doubtful
Human serum					
2	5	5	Not stated		
Blood transfusion					
5	3	3	Not stated		

The human serum was used subcutaneously in doses of from 30 to 60 cc. by one author in five acute cases, of which only

two recovered. It was used intraspinally, early by one author who did not comment on the result.

Blood transfusions are recommended by one author. Another reported improvement after their use. In one instance the patient died. Another patient died, but brain tumor was a complicating factor. The fifth author reported temporary improvement followed by death.

Summary comment.—There is no evidence to recommend the use either of normal human blood serum, or of blood transfusion, except in those cases in which blood transfusion may be indicated by the patient's general condition.

Bromides

There were a number of references to the use of bromides, but these were used so obviously for symptomatic treatment only, that it did not seem worth while to collect and correlate the references.

Bulbocapnine

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
9	45	45	Late	7	3

The drug was given orally or subcutaneously in doses of from 150 to 300 milligrams. There are ten references to comments. One author stated that the drug had no effect on cases of epidemic encephalitis, but did help cases of paralysis agitans. A second author reported that the drug had more effect on the tremor in other conditions than in Parkinsonism. Another author stated that bulbocapnine had more effect on tremor than did scopolamine, atropine or luminal. It was also

said that the drug was not habit-forming, and that increasing doses were not necessary. One observer noted that relatively small doses affected the perception and produced fatigue.

Summary comment.—There is some evidence that bulbocapnine is of value in controlling tremor. The reports are not unanimously favorable, however, nor are they sufficient to warrant definite conclusions at this time. This drug evidently has no specific effect on the encephalitic process.

For those interested in this drug the following references appear in the bibliography: F 55, J 54, J 55, J 56, J 57, J 58, L 244, M 252, S 82, E 38, and T 50a.

Caffein

There were a few references to caffein, sometimes used alone and sometimes in combination with quinine and strychnine, as well as with aspirin, phenacetin, bromides, chloral, and veronal. Its use was evidently purely symptomatic and the study of it was discontinued. Kolutrin, a caffein product from kola, was mentioned once.

Calcium

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
10	39	17	17	5	Early	1	2
					Late	1	3
					Not stated	3	

Calcium chloride was used intravenously in doses of from 5 to 10 cc. of a 10 percent solution. Calcium sandoz was given intravenously and intramuscularly. Calcium lactate was

given by mouth, and eusol, a combination of calcium borate, calcium chloride and hypochlorous acid, was given intravenously. This last was used in fifteen cases, in the acute stage, with unfavorable results.

Summary Comment.—The evidence is against the value of calcium, either in the early or the late stage of the disease.

Camphorated Oil

Work on the references to this drug was not completed, on account of its purely symptomatic use.

Cannabis Indica

Only one reference is given to this drug, for treatment of one late case, with unfavorable results.

Chloral Hydrate

As this drug was used only symptomatically, the study of the references was not completed. In general, its sedative effect seemed to be good.

Chlorinated Water

There was only one reference to chlorinated water. The number of cases treated was not given, and it apparently had no effect. It was said to have been injected, but neither the manner nor the amount was stated.

Cicutin and Conium

Number of references	Number of cases	Stage		Comments or results		
		Late	Not stated	Stage	Favorable	Unfavorable or doubtful
14	3	2	1	Late	1	3
				Not stated	4	2

The drug was given in the form of cicutine bromohydrate, subcutaneously, in doses of 1/2 to 3 mg.; also as fluid extract of conium in doses of from 3 to 15 mg. three times daily; and as conium hydrobromate.

Summary comment.—The evidence is entirely unsatisfactory, because of the limited number of cases and the uncertain results reported.

COLLOIDAL METALS

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
98	126 + "Many"	52 + "Many"	3	71	Early Late Not stated	23 7 26	8 18

Various colloidal metals were used. Electrargol was most frequently mentioned. Collargol, electrocollargol, colloidal gold, lantol (colloidal rhodium), colloidal mercury, colloidal iodine, orargol (10 percent gold, 90 percent silver), argotropan (1 percent colloidal silver, 20 percent hexamine), dispargen(a colloidal silver preparation), and other colloids were mentioned by trade names. Most of these colloidal preparations were given intravenously or intramuscularly. Electrargol was occasionally given intraspinally. Often it was simply stated that they were injected.

Summary comment.—As the basis of this treatment is entirely empirical, it is necessary that the evidence in support

of it should be unequivocal. This is not the case with colloidal metals. A much larger number of cases with adequate controls is necessary before definite conclusions can be drawn.

Convalescent Serum

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
62	102	76(?)	10(?)	16	Early	15	8
					Late	3	
					Not stated	10	14

The serum was used intraspinally in doses of 5 to 30 cc., intramuscularly in doses of from 20 to 80 cc., and intravenously in doses of from 50 to 100 cc.

Summary comment.—The evidence of results from this treatment indicate it has no value. In view of the chronicity of the disease, it is possible that the serum from apparently convalescent patients may still be infectious.

Curare

There was one reference to this drug citing three cases in the late stage. Curare was given intramuscularly in doses of 0.01, 0.02 and 0.03 cgr., with only temporary benefit.

Digitalis

As its use was purely symptomatic, work on the references was not completed.

ESERINE

Number of references	Number of cases	Stage Early	Comments or results		
			Stage	Favorable	Unfavorable or doubtful
5	1	1	Early	.....	1
			Late	.....	1
			Not stated	1	1

The eserine was administered hypodermatically in doses of 1/100 to 1/30 gr.; it was also given as eserine salicylate, 5 m. every six hours.

ETHER ANAESTHESIA

There was one reference to the use of ether anaesthesia, in one case in the early stage of the disease. The only benefit was the relief of myoclonus.

FIXATION ABSCESS

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
93	202	167	2	33	Early	21	9
					Late	.....	3
					Not stated	25	11

The fixation abscess was usually produced by the injection of from 1 to 2 cc. of turpentine. In some instances sterile pus from horses, injected with turpentine, was used instead.

Summary comment.—The relatively large proportion of unfavorable reports would seem to indicate that this mode of

treatment has little value. There is no evidence that fixation abscess exerts a specific influence on the course of the disease.

Gelsemium

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
7	18	.....	15	3	Early		
					Late	5	1
					Not stated	.....	1

The gelsemium was administered as the fluid extract, 7 m. orally, or as the hydrochlorate, 1/30 gr. orally or subcutaneously, three times a day. It was also stated that 1/7 mg. of gelsemium was given.

Summary comment.—The evidence is insufficient.

Gentian Violet

Three early cases, treated with gentian violet, were referred to, in one reference; the outcome was stated to be favorable. Gentian violet was given intravenously, 3 mg. per kilogram of body weight. The evidence, however, either for or against this drug is insufficient.

Glucose

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
7	3+ "Several"	3	"Several"	Early	.....	3
				Not stated	4	

Glucose was given intravenously in doses of varying sizes, from 30 to 300 cc. of a 25 percent solution to 500 cc. of a 10 percent solution. Adrenalin was added in one instance.

Summary comment.—Evidence available in reports is inconclusive. Verbal reports from authoritative sources are favorable, although no specific value is claimed.

Gomerol*

Gomerol was administered to one patient, who recovered. The size of the dose and the method of administration were not stated.

Hyoscine

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
88	78	3	65	10	Early	3	
	+		+		Late	50	10
	"A few"		"A few"		Not stated	13	2

Hyoscine was usually administered in the form of hyoscine hydrobromide, hypodermatically in doses of from 1/400 to 1/100 gr. It was also given orally, 1/25 to 1/100 gr. It was sometimes combined with other drugs, as stramonium, belladonna, scopolamine, chloral hydrate, or codeine sulphate. Duboisin, an alkaloid identical in composition with hyoscyamine, was mentioned four times. It was given orally in doses of from 2 to 3 mg. daily.

Summary comment.—Evidence shows hyoscine to be one of the most reliable drugs in the symptomatic treatment of epi-

* A complex oily liquid distilled from the leaves of niavul.

demic encephalitis. It acts in the early stage to quiet delirium and mental excitement, and to arrest myoclonic twitchings. It is especially useful in late stages to control tremor, especially of the Parkinsonian type, and to lessen the intensity of myoclonic and ocular spasms. There is no evidence that it has a specific influence on the process of the disease.

IODIDES, TOTAL

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
99	291	144	127	20+ "Several"	Early	20	5
					Late	23	12
					Not stated	16	6

A great variety of iodine preparations were used: tincture of iodine administered by mouth; potassium iodide; iodoform; septo-iodine; Pregl's solution given intravenously, in doses of from 10 to 300 cc.; sodium and potassium iodide with bromides; iodine and arsenic; sodium iodide glucosated with arsenic, given intravenously; sodium iodide given intravenously, in doses of 100 cc. of a 10 to 25 percent solution. One writer used sodium iodide intraspinally and also in the ventricle.

Summary Comment.—The evidence is insufficient to justify final conclusions. Enthusiastic recommendations for intravenous administration have been made. The justification of claims of benefit from this treatment is not yet established. Evidence regarding a specific influence of iodides on the lesions is not convincing. This method requires extensive further trials, properly controlled.

IODIDES INTRAVENOUSLY INJECTED

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
Iodoform 1	2	.....	.....	2	Not stated	1 (somewhat favorable)	
Septo-iodine* 2	2	1	.....	1	Early Not stated	1 1 (improved ocular spasms)	
Pregl's solution† 10	100	11	21	68+ Several	Early Late Not stated	5 2 2	3
Sodium iodide‡ 9	64	2	62	.....	Early Late	 7	2

* Dose, 10 cc.

† An antiseptic solution of the sodium salt of hydriodic acid and iodic acid with metallic iodin, 0.04 per cent. Dose, 10 to 300 cc.

‡ Dose, 100 cc. of a 10 to 25 percent solution.

On account of the interest in the intravenous use of iodides this detailed table of references to this method of treatment has been included.

LUMINAL AND GARDENAL*

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
28	29	2	15	12	Early	2	
					Late	12	4
					Not stated	3	1

* Gardenal is a French preparation of luminal.

Luminal was usually given by mouth in fairly large doses, sometimes as large as 6 grains daily. In one instance a combination of luminal and sodium was given intraspinally. Gardenal was usually given by mouth, 10 to 20 cgr. daily. It was also given by injection, and sometimes in combination with arsenic or hyoscine.

Summary Comment.—The evidence indicates it to be of some value in symptomatic treatment.

MAGNESIUM

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
15	9	3	6	Early	.....	1
				Late	2	
				Not stated	4	4

Magnesium was used as the sulphate, except in one instance, when the chloride was used. Magnesium sulphate was administered intraspinally; also intravenously, in doses of from 4 to 10 cc. of a 25 percent solution; intramuscularly, in doses of 5 cc. of a 25 percent solution; and also as a high enema combined with camphor and tincture of opium.

Summary Comment.—There was no evidence of any beneficial effect.

Malaria Inoculations

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
9	24	24	Late	1	5

Summary Comment.—The weight of evidence is against the use of the malarial plasmodium in the treatment of epidemic encephalitis.

Medinal

The use being symptomatic, work on references was not completed. It was apparently a satisfactory sedative and hypnotic.

Mercurochrome

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
5	5	2	3	Early	1	1
				Not stated	1	2

The mercurochrome was used intravenously, 5 mg. per kilogram of body weight in a 1 percent solution. It was used intraspinally in one case, which had a fatal outcome, the dosage being 2-5 cc. of a 0.5 percent solution.

Summary Comment.—The weight of evidence is against the use of mercurochrome, but the number of cases is too small to make the evidence of value.

MERCURY

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
47	73	29	25	19	Early	7	2
					Late	5	6
					Not stated	14	4

The mercury was given at times by inunction. Various compounds were used, such as bichloride, cyanide, benzoate, salicylate, gray powder, calomel, biniodide, and enesol (mercury salicylarsenate), which was given intramuscularly, in doses of from 3½ to 30 cc., and intravenously, in doses of from 5 to 10 cc. The cyanide of mercury was also given intravenously in some instances.

Summary Comment.—There is no evidence to show that mercury exerts any specific control over the process or lesions of epidemic encephalitis.

METHYLENE BLUE

Methylene blue was used once, and argochrome (a combination of silver and methylene blue) once. Neither the dose nor the method of administration was stated.

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
2	2	1	1	Early	1	
				Not stated	1	

Summary Comment.—The evidence is insufficient.

Miscellaneous Foreign Protein

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
50	117	55	50	12	Early	6	2
					Late	13	6
					Not stated	10	5

A great variety of foreign proteins were used, often called by trade names, and sometimes it was merely stated that protein therapy was employed. Intramuscular injections of from 2 to 10 cc. of sterile milk were perhaps most frequently used. Casein was used intraspinally, 0.5 to 2 mg. in 10 cc. physiological salt solution; buttermilk was also injected intraspinally. Peptone and leucotropine were injected intravenously. Typhoid vaccine was mentioned four times; the dosage in one instance was from 2 to 20 million bacilli intravenously.

Summary Comment.—The evidence concerning the value of treatment by foreign protein shock is not decisive with regard to any specific action upon the disease. The number

of unfavorable reports bear about the same ratio to the favorable, i.e.: 1:2, or 1:3, that has existed in regard to many of the therapeutic agents already considered.

NICOTINE

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
4	30	30	Late	4	

The nicotine was given subcutaneously in doses of 1/30-1/5 gr.

Summary Comment.—There is some evidence that nicotine is useful in relieving the tremor, but it may have a tendency to increase the rigidity. This objection makes its usefulness questionable. This drug has nothing more to recommend it than has hyoscine and its derivatives.

NOVOCAIN

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
2	7	7	Late	1	
			Not stated	.....	1

The novocain was given in the epidural space in one instance, and by injections in another, the dosage being 5 to 8 cc. of an 0.5 percent solution.

Summary Comment.—The evidence is too limited for any conclusions to be drawn.

Opium

The references to this drug were apparently to its symptomatic use only. The work on the references was therefore not completed.

Organotherapy

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
52	29	5	16	8	Early	4	1
					Late	7	8
					Not stated	6	9

Various endocrine preparations were used, and often a combination of several, so that an exact classification is not easily made. Some preparation of the pituitary gland, either alone or in combination, was referred to nineteen times; thyroid preparations eleven times; parathyroid four times; suprarenal three times; an ovarian product once; testicular extract once; Steinach's operation was performed once, and there was a transplantation of the parathyroid, without success.

Summary Comment.—Except in obvious cases of glandular disfunction, organotherapy is evidently of no value.

Oxygen

The oxygen was used by inhalation by one author; by another, both by inhalation and in subcutaneous injections of

Number of references	Number of cases	Stage		Comments or results		
		Late	Not stated	Stage	Favorable	Unfavorable or doubtful
2	10	8	2	Late	1	1 (case with tuberculosis)
				Not stated	.....	1

300 cc., combined with subcutaneous injections of potassium permanganate.

Summary Comment.—This treatment has little to recommend it, either empirically or scientifically.

Paraldehyde

Number of references	Number of cases	Stage		Comments or results		
		Early	Late	Stage	Favorable	Unfavorable or doubtful
9	6	1	5	Early	1	
				Late	1	
				Not stated	6	

The paraldehyde was given either orally in doses of from 1 to 4 drachms, or by rectum in doses of from ½ to 1 ounce. Its use was evidently symptomatic.

Phenol

One reference was made to phenol treatment, in which 1 cc. of a 3 percent solution was given along the spine intradermally, with favorable results.

Summary Comment.—This treatment seems entirely without logical foundation.

PHYSIOTHERAPY

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
*Electrotherapy**							
28	8	2	3	3	Early	1	1
					Late	5	3
					Not stated	2	3
Radiotherapy							
20	58	1	56	1	Early	1	
					Late	8	3
					Not stated	3	1
X-ray							
11	9	0	4	5	Early		
					Late	1	4
					Not stated	6	
Hydrotherapy							
14	2	0	2	0	Early	1	1
					Late	4	2
					Not stated	2	0

* Galvanic, faradic and sinusoidal currents were used, also diathermy.

General exercise, swimming and reëducation of muscles were referred to several times, with generally favorable comments.

PICROTOXIN

One reference was given to the use of picrotoxin in a case in the late stage, in which the result was doubtful. The picrotoxin was administered orally and subcutaneously, 1/16 gr. three times a day.

Summary Comment.—This treatment has no logical foundation.

Potassium Permanganate

Number of references	Number of cases	Stage		Comments or results		
		Early	Late	Stage	Favorable	Unfavorable or doubtful
3	9	1	8	Early	.....	1
				Late	1	(Cases with tuberculosis made worse)
				Not stated	1	

Potassium permanganate was used by one author subcutaneously, in a dose of 1 cc. of a 1 percent solution, combined with oxygen subcutaneously. This has already been referred to under Oxygen. In one instance it was administered by rectum and in another it was used for disinfection of the nasopharynx.

Summary Comment.—There is no evidence from which to draw any conclusions in regard to the value of potassium permanganate in this connection. Its power as a disinfecting agent is well recognized.

Psychotherapy

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
25	20	0	3	17	Early		
					Late	3	5
					Not stated	7	6

Hypnosis, suggestion, psychoanalysis, and psychotherapy, were mentioned.

Pyramidon

Four references were given to the use of pyramidon. Two gave favorable results; the stage of the cases was not, however, stated. One reported unfavorable results, and one author did not comment on the result. Pyramidon was combined with phenacetin, and also with stovain and veronal. Its use was apparently only symptomatic.

Quinine

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
51	72	26	46	Early	9	3
				Late	.....	1
				Not stated	15	12

Quinine was usually administered orally; sometimes intramuscularly, intravenously, or rectally. It was sometimes combined with hyoscine, bismuth, caffeine and strychnine, or urotropine and sodium salicylate. Various compounds were used, such as optochin, vuzin, eucupin, and diphenylethylhydrocuprein.

Summary Comment.—The evidence shows little in favor of quinine. It seems to have no specific effect on the disease.

Salicylates

Sodium salicylate was the preparation most often used, but sodium bisalicylate, aspirin and salol were also mentioned. Sodium salicylate was given orally, sometimes in combination with urotropin and ammonium benzoate, urotropin and strych-

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
96	284	210	38	36	Early	34	7
					Late	11	5
					Not stated	19	14

nine, urotropine and quinine, urotropin, postassium iodide, or sodium cacodylate. Sodium salicylate was also given intramuscuarly, intraspinally and intravenously. When given intravenously, a solution in 10 percent glucose was often used to prevent the obliteration of the veins. The intravenous dose was usually from 7 1/2 to 45 grains.

Summary Comment.—The salicylates seem to maintain their reputation for universal service in the treatment of a wide range of symptoms.

SALT SOLUTION

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
17	20	2	18	Early	1	1
				Not stated	8	

The salt solution was usually employed as a hypodermoclysis, the dosage being from 200 to 500 cc. of normal saline, or when given intravenously, 500 cc. Adrenalin was used with the hypodermoclysis in one instance. Ringer's solution was used twice as a hypodermoclysis, 200-300 cc. twice a day,

and 1000 cc. twice a week. In two instances, 10 cc. of a 15 percent hypertonic salt solution was used intravenously. One author noted that the hypertonic salt solution was not so good as a hypertonic glucose solution.

Summary Comment.—The evidence indicates that there is no specific value in the use of salt solution. Its value for symptomatic relief as a hyperdermoclysis is self-evident. As a hypertonic solution, it is evidently inferior to hypertonic glucose.

Salvarsan Preparations

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
73	75	33	5	37+ "Several"	Early	11	4
					Late	3	3
					Not stated	21	18

Salvarsan, arsphenamin, or arsenobenzol were mentioned sixteen times; neosalvarsan, neoarsphenamine, novarsenobillon, and novarsenobenzol much more frequently. Silversalvarsan and neokharsivan were each mentioned in two of the references.

Summary Comment.—The value of salvarsan or allied arsenicals is doubtful.

Scopolamine

Scopolamine was given orally and hypodermatically. The dosage was usually from ¼ to 1 mg. It was at times combined with sodium cacodylate, gardenal, morphine, or hyoscine, and alternated with atropine. Genoscopolamine was

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
131	109	12	84	13	Early	4	2
					Late	70	12
					Not stated	27	1

mentioned as having a more lasting effect, and as being less toxic than hyoscine hydrobromide.

Summary Comment.—For the symptomatic relief of tremor, scopolamine ranks about the same as hyoscine.

Sera*

Anti-encephalitis serum. (Horse, immunized against polioencephalitis in horses.) There is one reference to this serum. It was used subcutaneously, intravenously and intraspinally in two early cases. The result is not stated.

Anti-encephalitis serum (Freeman). A horse was immunized with strains of organisms isolated from cases of encephalitis by Evans and Freeman.

There are two references to the use of this serum, both by Freeman. Fourteen cases were treated, nine acute, four chronic and one with stage unstated. It was used subcutaneously or intravenously, in doses of from 50 to 100 cc. While it was stated that the results were favorable in some cases, apparently equally favorable results were obtained with a nonspecific serum. The latter fact, together with the small number of cases treated, leaves the value of the specific serum in doubt. There is nothing in the results to recommend the use of this serum.

* With claims of specificity.

Rosenow's anti-encephalitis serum.—The serum was injected intramuscularly, intravenously and intraspinally in doses from 5 to 40 cc. In regard to the largest series of cases, 130 in all stages, it is stated that there was improvement in 65 percent of cases, mostly in the acute stage, and also some improvement in the chronic cases.

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
11	155	25	130 all stages	Early	3	3
				Late	1	
				Not stated	.....	2

Summary Comment.—Figures on the basis of cases treated and reported as improved, show about the same proportion of favorable results as untreated cases. It is also to be noted that of the total number of comments made, more are unfavable. There is nothing to indicate that this serum is specific for encephalitis.

Sera, Non-Specific

In addition, two other sera were each mentioned once, Query's serum and anti-pyogenic serum. No description of the latter was found. Query's serum, obtained from a monkey immunized by injection of the spirocheta pallida, was injected in one patient, who recovered; the anti-pyogenic serum was injected (manner and dose not stated) in four early cases. The results were not favorable.

Summary Comment.—The variety and number of sera used and their complete lack of relation to the etiology of the dis-

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
*Normal horse serum**							
10	9	0	0	9	Early	1	
					Not stated	5	1
*Antidiphtheritic serum***							
22	16	11	0	5	Early	6	1
					Not stated	8	2
Polyvalent grippe serum†							
11	40	36	1	3	Early	2	2
					Late	1	
					Not stated	2	2
Antimeningococcic serum††							
17	22	20	0	2	Early	3	4
					Not stated	3	
Anti-pest serum‡							
2	2	1	0	1	Early	1	
					Not stated	.....	1
Anti-pneumococcic serum‡‡							
5	7	1	0	6	Early	1	
					Not stated	3	
Anti-streptococcic serum§							
8	13	12	0	1	Early	1	
					Not stated	2	2
Anti-tetanus serum§§							
10	10	2	2	6	Early	1	
					Late	1	
					Not stated	5	1

ease, renders any observations upon them of little scientific value. It is significant to note that the majority of comments are favorable in regard to these sera, which can have no claim to specificity. The evidence concerning the results obtained from this form of therapy does not justify any conclusions in its favor.

SIALOGOGUES*

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
34	25	10	7	8	Early	3	
					Late	2	3
					Not stated	19	1

* Pilocarpine and jaborandi.

* The serum was given subcutaneously, intramuscularly, intravenously and intraspinally, in doses of 10 to 20 cc.

** The serum was used subcutaneously or intravenously in doses up to 20 cc. and subdurally, the only dose stated being 5 cc.

† Grippe serum; influenza serum; antipheiffer serum; given subcutaneously or intramuscularly in doses of from 5 to 40 cc., and intraspinally, in doses of from 5 to 30 cc.

†† The serum was given subcutaneously and intramuscularly, but usually intraspinally, in doses of 10 to 50 cc.

‡ The serum was injected, route not stated, in doses of 20 cc.

‡‡ It was stated once that the serum was used intraspinally. The other references did not give the method.

§ The serum was injected subcutaneously and intravenously in doses of 10 to 20 cc. and once intraspinally.

§§ The serum was injected intraspinally and intramuscularly—the doses were not stated.

Pilocarpine was used more frequently than jaborandi. It was administered by instillation in the eye, orally, and by injections. Jaborandi was sometimes given in the form of an infusion. Both were sometimes combined with adrenalin.

Summary Comment.—Value only for symptomatic relief is claimed for these drugs.

SODA

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
7	1+ "Several"	1	"Several"	Early	1	
				Late	2	1
				Not stated	2	1

The formiate of soda was referred to twice; the nucleinate, three times; the citrate, once; and Hayem's serum, once. The sodium was given intramuscularly; the sodium citrate intravenously, in a 2 percent solution. The dosage was not stated.

Summary Comment.—The evidence is inconclusive.

SOLANIN

There is one reference to solanin. Neither the dosage nor the method of administration is stated. It was used in late cases, and was said to be not always effective.

STRAMONIUM

The stramonium was usually given in the form of pills of the powdered leaf (datura stramonium) in increasing doses, up to 1 to 2 grams per day. Some authors suggested a rest for several days, after ten days' treatment. The tincture of

Number of references	Number of cases	Stage		Comments or results		
		Late	Not stated	Stage	Favorable	Unfavorable or doubtful
15	58	58	1	Early	1	
				Late	10	
				Not stated	2	

stramonium in doses of 20 to 90 mg. per day was also used. In one series, the effect was compared with laevo- and dextro-rotating hyoscine, and with atropine. It was found that better results were obtained with stramonium. The improvement consisted in a lessening of the muscular rigidity and over-salivation, and an improvement in posture, speech and mental condition. The toxic effects were blurring of vision, which one author treated with eserine, and dryness of the mouth, treated by adding tincture of jaborandi and by the use of Burroughs and Wellcome's "thirst quencher" tablets.

As stramonium is receiving rather favorable comments in recent articles, references to the bibliography are appended: J 72a, J 72b, J 73, L 37a, L 37, L 38, L 231, L 81, M 162, P 224, R 30a, S 161a, S 408, C 38a, G 128.

STRYCHNINE

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
44	52	26	15	12	Early	11	2
					Late	3	2
					Not stated	11	3

Strychnine was often given by hypodermic injections. It was sometimes combined with caffeine and quinine, urotropine, adrenalin, urotropine and sodium salicylate, or sodium cacodylate. Strychnine cacodylate, with or without arsenate of iron, was mentioned once. Tincture of nux vomica was occasionally used.

Summary Comment.—Its use was apparently only for symptomatic relief.

SULPHUR

Number of references	Number of cases	Stage	Comments or results		
		Late	Stage	Favorable	Unfavorable or doubtful
2	9	9	Early Late	1 2	

One author stated that a 1 percent solution of sulphur depurata was injected intramuscularly, the dosage beginning with 1 cc. and increasing. He reported definite improvement in nine late cases and stated that the results in early cases were unfavorable. The second author recommended sulphur preparations in late cases.

SURGERY

Number of references	Number of cases	Stage		Comments or results		
		Early	Late	Stage	Favorable	Unfavorable or doubtful
5	70	3	67	Early Late	1 5	

The procedures that might be classed as surgical, were freezing with ethyl chloride, or severing the phrenic nerve; washing out the sinuses; removal of foci of infection, as tonsils or teeth; and sympathetic ramisectomy. This last operation was performed on sixty-two patients. The results of the first two were reported as favorable. Results on the remaining sixty have not been published, so far as we have been able to determine.

TARTAR EMETIC

Number of references	Number of cases	Comments or results		
		Stage	Favorable	Unfavorable or doubtful
2	Not stated	Early	1	
		Late	1	
		Not stated	.....	1

One author used tartar emetic intravenously without results. The second used stibiated tartar intravenously, 2 to 10 cc. of a 10 percent solution. He reported recovery in early and late cases, but did not state the number of cases.

THEOBROMINE

Theobromine was used by one author in one case, the stage of which was not stated. It was given in 1 gr. doses, route not stated, with favorable results.

TURPENTINE

Turpentine was used extensively for the production of fixation abscesses and has already been mentioned under that heading. In these cases it was apparently not used for that

Number of references	Number of cases	Stage		Comments or results		
		Early	Not stated	Stage	Favorable	Unfavorable or doubtful
3	3	1	2	Early	.....	1
				Late	1	
				Not stated	.....	1

purpose, although two articles refer to its use subcutaneously. In one account the essence of turpentine was used intraspinally in an acute case which resulted fatally.

Urethane

There was one reference to urethane, used in one early case intramuscularly, in 40 mg. doses. The result was favorable.

Urotropine

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
331	708	234	42	432	Early	56	35
					Late	10	10
					Not stated	101	56

We find in the references accounts of the use of urotropine, hexamine, hexamethyleneamine, hexamethylenetetramine, uroformin and methenamin. The drug was given alone, also in combination with sodium salicylate and ammonium benzoate; with salicylate; with salicylates and strychnine; with adrenalin; with salol; with chlorine; with quinine; with quinine and

salicylate; or with acid sodium phosphate. It was administered orally in doses of from 5 to 20 gr. three times a day; subcutaneously or intramuscularly up to 15 grains; intravenously in doses of from 15 to 45 grains in normal saline; intraspinally from 15 to 30 grains, made up in a 40 percent solution; by enemata, and often by combination of several of these methods.

Summary Comment.—For some reason this drug was used more extensively than any other. It has no specific influence on the disease. The claim made that it appears in the spinal fluid as formalin has not been substantiated. The comparatively large number of unfavorable comments is significant. There is a suspicion that it may have been used by force of habit or as a placebo.

VACCINES (BACTERIAL)

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
*Influenza-antigrippe**							
11	39	25	.....	14	Early	6	1
					Not stated	1	
Autogenous vaccine							
4	.....	Few	.....	.....	Early	1	
					Late	2	1
					Not stated	1	
Miscellaneous bacterial vaccines							
15	21	5	6	10	Early	3	1
					Late	.....	1
					Not stated	6	2

* The influenza vaccine was given usually subcutaneously; once intravenously

Summary Comment.—There is nothing in the evidence to recommend these vaccines.

VACCINE (RABBIT BRAIN, LEVADITI)

Number of references	Number of cases	Stage			Comments or results		
		Early	Late	Not stated	Stage	Favorable	Unfavorable or doubtful
14	99	16	65	18	Early	3	
					Late	5	2
					Not stated	1	1

This vaccine is prepared from the brains of rabbits inoculated with Levaditi's strain "C." The brain is emulsified in normal saline. It was at first used intraspinally, 1 to 5 cc. of a 5 percent emulsion. It is now used by subcutaneous or intramuscular injection. Most of the reports given in the literature are on the results obtained by intraspinal injection.

Summary Comment.—If Levaditi's strain "C" is the etiologic agent of epidemic encephalitis, this treatment rests upon a scientific basis. Many of the comments are favorable, but the data are as yet insufficient to enable us to reach definite conclusions.

A list of references to the bibliography is appended: D 179, G 58, G 116, H 113a, H 140, L 103, L 175, M 85, M 87, M 88, N 102, P 177, S 401a.

Chapter IV

EPIDEMIOLOGY

Historical Account

There is no record of a previous outbreak of any similar disease comparable in extent and severity to the recent appearance of epidemic encephalitis. Many writers, however, have endeavored to show a relationship between the disease as we now know it and such earlier epidmics as the Tübingen *Schlafkrankheit,* the Italian *nona,* and others. Crookshank (C 280a) in his book on "Influenza" has given a complete analysis of the historical evidence with regard to those events.

The indeterminate characer of epidemic encephalitis renders conclusions concerning the nature of these previous epidemics uncertain. Two recent outbreaks of considerable importance—the Australian X-disease of 1917-18, and the Japanese encephalitis epidemic of 1924—have received careful study, but their relationship to epidemic encephalitis remains uncertain. The diagnosis of a condition that occurred thirty or a hundred years ago must be even more difficult.

The first recorded cases of this epidemic had their onset in April and May, 1915, in Bucarest, Rumania, and were reported in 1916 by Obregia, Urechia and Carniol (O 6a, U 7) as "Encephalite hemorrhagique avec un diplocoque encapsulé."

Shortly afterward, on April 27, 1917, Cruchet, Moutier and Calmettes (C 314) published their article on "Quarante cas d'encéphalo-myélite subaiguë." These forty cases (later accounts added to the number) were studied in the French military hospitals, and occurred at Commercy, Verdun and Bar-le-duc in 1915 and 1916. Etienne's cases (E 57, E 58) which

also occurred in the French army in August, 1915, and May, 1916, were in all probability epidemic encephalitis and should be included here.

The group observed by von Economo (E 11, 11a, 13) in Vienna came later, in January and May, 1917. As profound lethargy was an outstanding clinical feature in these cases, von Economo applied the term encephalitis lethargica. This name was at once adopted and was used widely at first. Later, however, it became evident that lethargy was an inconstant symptom and the name given by Economo was gradually supplanted by the more general term epidemic encephalitis.

At about the same time as von Economo's observations, sporadic cases appeared in widely separated districts. In Pola, on the coast of the Adriatic, a cadet was taken sick on February 16, 1917 (R 289); it is perhaps worthy of note that he had been in Vienna on leave February 3-12. In Kiel, Germany, one case developed in February (R 59), and another in Berlin, in May (K 56a); in February, there was a case in Warsaw, Poland (S 318); one in April in Kladno, Czechoslovakia (P 214). Early in 1917 also came the first reports of the X-disease in Australia. A case was observed in Morocco in June (P 224). In November and December, three cases were reported from Manchester, England, as epidemic polio-encephalitis (so-called epidemic botulism) (M 253); and in December, a case occurred in Basle, Switzerland (B 225, S 300).

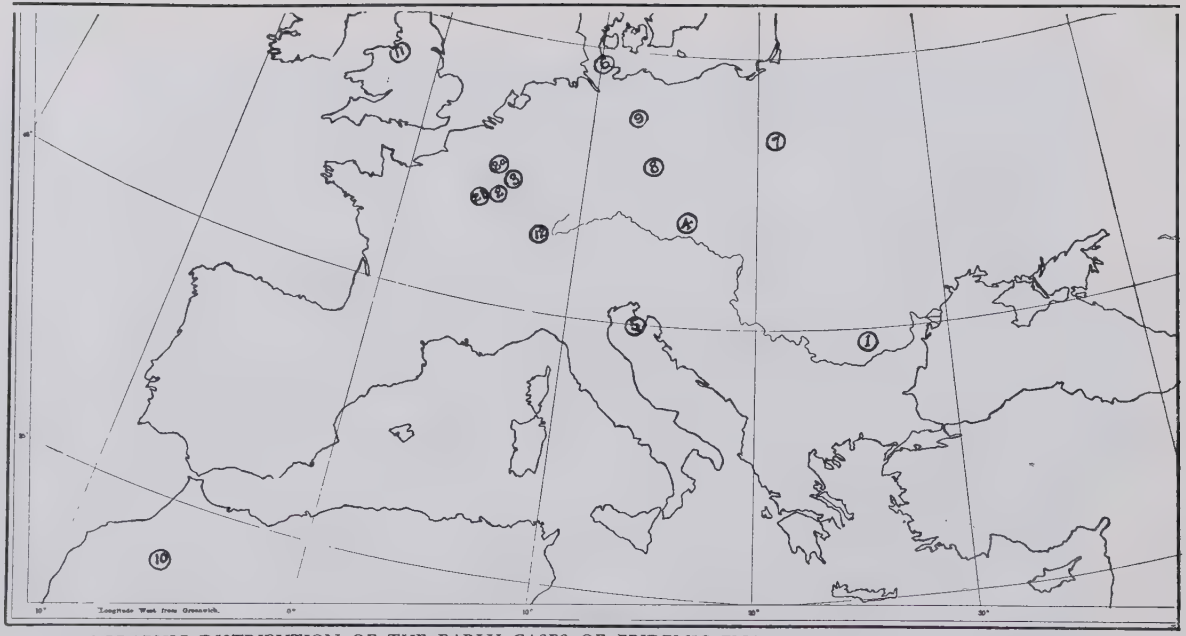
Even during 1915 and 1916, there were scattered cases in many countries, which were unrecognized in the acute stage but whose true character became apparent later, when a chronic condition developed. Among these may be mentioned Hall's case in England, June, 1916; Moewes' case in Berlin, April, 1915; and Watson's case in Yunnanfer, China, in 1915.

A study of the literature prior to 1915, reveals still other cases that, in view of our present understanding, may be looked upon as sporadic cases of epidemic encephalitis. Hall (H 29a) quotes several such instances: one described by Raymond and Claude in 1909 as "serous meningitis with fever, somnolence and diplopia; at autopsy perivascular cuffs were found in the central nervous system"; another, a case of paralysis agitans dating from a probable attack of encephalitis in 1910, reported by Souques; and two cases of "focal encephalitis" recorded in 1912 by G. W. Watson.

Tilney and Howe (T 60) describe a case of the lethargic type, with onset in the winter of 1912-13. Serial sections of the entire brain stem were obtained for study.

Even now, from time to time, patients are discovered who are suffering from chronic conditions probably following an acute attack of epidemic encephalitis dating back to pre-epidemic years. The history of such a case is given by Dr. Neal (unpublished data). This patient, born in July, 1910, developed normally up to the age of eighteen or twenty months. He had learned to walk and to talk. He had had no infectious diseases. During the winter of 1911-12 he was ill with a fever, was stuporous and had convulsive movements. He became unable to walk or to talk and was in bed for six years. The parents had been told that he had poliomyelitis, a diagnosis based evidently on the inability to walk. When seen in July, 1928, he was mentally deficient, had an ataxic gait and general athetoid movements. The diagnosis of a post-encephalitic condition was confirmed on consultation with Dr. Hubert Howe.

Green (G 128) has recently described a case of Parkinsonism following an attack of acute encephalitis in 1914.



GEOGRAPHIC DISTRIBUTION OF THE EARLY CASES OF EPIDEMIC ENCEPHALITIS, 1915 to 1917 (Key to Map)

(1) Bucarest, Rumania, 2 cases, April and May, 1915 (O 6a, U 7)
(2) Commercy, France, 2 cases, February and September, 1915
(2a) Verdun, France, 5 cases, Winter of 1915-16
(2b) Bar-le-Duc, France, 48 cases, March, 1916-May, 1917
(C 309, C 313, C 315)
(3) French Military Front 2 cases, August, 1915, 10 cases May, 1916 (E 57, E 58)
(4) Vienna, Austria, 13 cases, January-May, 1917 (E 11, E 11a, E 13)
(5) Pola, Province of Istria, Austria, (now part of Italy), 1 case, February, 1917 (R 289)
(6) Kiel, Germany, 1 case, February, 1917 (R 59)
(7) Warsaw, Poland, 1 case February, 1917 (S 318)
(8) Kladno, Czechoslovakia, 1 case, April, 1917 (P 214)
(9) Berlin, Germany, 1 case, 1917 (K 56a)
(10) Morocco (exact location not given), 1 case, June, 1917 (P 224)
(11) Manchester, England, 3 cases, November-December, 1917 (M 253)
(12) Basle, Switzerland, 1 case, December, 1917 (B 225, S 300)

Geographical Distribution

1918.—Early in 1918, a considerable group of cases of epidemic encephalitis developed in England; others were reported from Glasgow and Dublin; one from Algiers; and three from Florence, Italy. The second outbreak of the X-disease in Australia reached its peak in February. Netter saw seven cases in Paris in March, and others were reported from Bordeaux in March and April. Professor Morquio wrote to Netter describing three cases seen in Uruguay in August, 1918. The French writer also describes two cases seen by Dr. Caryophyllis in Greece in October. Two cases were reported from Morocco late in the year. Sporadic cases occurred also in Sweden, Belgium and Holland. In Germany, the total number of cases was small for the year, but they were widely distributed. Hoff reported 174 cases in Vienna in 1918. In September and October the disease was observed in the United States, in New York City, Iowa and West Virginia.

1919.—In 1919 epidemic encephalitis was again prevalent in England and France. The first epidemic increase in Germany and Austria was observed during this year. There was a slight accumulation of cases in Holland and Belgium; small groups appeared in Norway and Sweden in the summer and fall. A few cases were seen in Portugal, and the end of the year marked the beginning of the epidemic in Spain. In China, Cadbury's case occurred in February. Three cases were reported from South Africa in February and March; and ten from New Zealand (R 264) during the period from March to May. Two cases were observed in Sicily (L 272) in January and February. In Havana, there were two cases in March (M 361); and in May sporadic casese were seen in Argentina, Brazil and Peru. In the autumn the disease appeared in

Canada, the Philippines, the Ukraine, Western Siberia and India. One case (H 27) occurred in Mesopotamia in December (retrospective diagnosis), and another in Peking.

1920.—Late in 1919, there was an increase in the number of cases of epidemic encephalitis in Poland, Switzerland and Italy; this marked the beginning of the 1920 pandemic, the manifestations of which were especially violent in these three countries. In Czechoslovakia, too, the epidemic began in December, 1919, but did not become pronounced until January, 1920. In Austria the peak was reached in 1920. There was a considerable increase in the number of cases in Germany. Official reports showed a slight increase also in England, in Holland and in Denmark. In France, the presence of a severe epidemic was first recognized during this year. Spain and Portugal were also affected. The disease became established in Greece, and a group of cases was reported in Sicily (L 272). The United States and South America shared in the general outburst. Groups of cases were reported from Central Russia, from China, and from South Africa. Isolated cases were seen in Bagdad (G 115), in Alexandria (V 3) and in Palestine (David, J., *Bull. et mém. Soc. méd. d. hôp. de Paris,* 44: 493, 1920).

1921.—Although there was a general decline in the severity of the epidemic during 1921, this year marked its peak in Norway and Sweden, in Finland and in Belgium. There was a serious outbreak in England at the end of 1920 and during the first quarter of 1921. In France the disease occurred in an especially violent form in January (E 43). There was a small epidemic in Southern Lappland (Sweden) in February. In March one case was reported from the Isthmian Canal Zone (B 393).

1922 and 1923.—The world-epidemic reached a low level

during 1922. Several groups of cases developed, however, during the winter of 1922-23. The second Winnipeg epidemic occurred at this time. In the Philippines, the disease made its first appearance in epidemic form in November, 1922, and in the spring of 1923. There was a small epidemic in Moscow during this same period. In Western Siberia the height was reached between 1922 and 1924. In 1923 there was a renewed outbreak in Poland and this year marked the peak of the epidemic in Canada, and in the United States. Seven cases were observed in the Samoan Islands in the spring of 1922 (M 166); and ten in Syria between December, 1922, and April, 1923 (S 173). In November, 1923, a case was seen in Dakar (Cape Verde) (H 113).

1924.—The second peak of the pandemic of epidemic encephalitis occurred in 1924. During this year, the incidence in England, Wales, Scotland, and Ireland reached its height. Official reports for Prussia were also highest at this time Of the cases observed at the Union Medical College, China, the greatest number for any one year occurred in 1924. This year also was notable for the great epidemic in Japan. The nature of the disease observed there and its relationship to epidemic encephalitis is discussed in the section on Japan.

1925, 1926, 1927 and 1928.—No important outbreaks have occurred since 1924. "Encephalitis lethargica seems to have lost its epidemic character, at least for the time being. Its incidence has decreased in all countries in recent years and its seasonal fluctuation has almost disappeared. . . .

"It appears from statistics for the first half of 1928 that the general decrease of encephalitis lethargica is still continuing in most countries. In Prussia and Scotland, however, the incidence was the same as during the first half of 1927,

and there was a slight increase in Sweden" (*League of Nations Monthly Epidemiological Report,* R. E. 116, July, 1928).

The *League of Nations Monthly Epidemiological Reports* show that cases of epidemic encephalitis have been reported from time to time during the recent epidemic period in widely separated portions of the world. These case reports have been collected in Table I, which is not offered as representing a complete record of the incidence of epidemic encephalitis in the countries mentioned, but simply as an indication of the fact that the disease is not limited to any geographical area.

Table I.

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN CERTAIN MINOR GEOGRAPHICAL DIVISIONS

		1920	1921	1922	1923	1924	1925	1926	1927
Argentina		4							
Brazil		8							
Cyrenaica		....	....	....	....	1	0	0	0
Esthonia		....	....	0	12	3	6	1	3
Jamaica		....	....	....	....	....	1	....	1
Kingdom of the Serbs	C	....	....	....	....	....	37	15	22
Croats & Slovenes	D	....	....	....	....	....	12	9	11
Latvia		....	....	3	0	3	1	5	0
Lithuania	C	....	....	0	72	7	2	4	6
	D	....	....	0	7	1	0	0	0
Luxemburg		4	....	....	....	....	1	0	0
Malta		3	8	2	0	40	26	12	13
Saar Territory	C	....	....	....	....	4	13		
	D	....	....	....	....	3	4		
Syria and the Grand Lebanon		....	....	....	....	....	....	12	18
Turkey		....	....	....	....	0	4		

From the *League of Nations Monthly Epidemiological Reports.*

World Incidence

To compile an accurate statement of the total world incidence of epidemic encephalitis during the past ten years is impossible. In some countries the disease was not made notifiable until the peak of the epidemic was past. Moreover, notification of this disease "is undoubtedly imperfect in all countries" (*League of Nations Monthly Epidemiological Report,* R. E. 116, July, 1928). "For every 100 cases notified there are about 50 to 75 mostly mild cases which escape notification" (P 46).

We give below, for comparison, two sets of figures. In the first column (taken from the *League of Nations Monthly Epidemiological Reports*) the results were obtained by "adding together the returns for six European countries for which fairly reliable data are available since 1920 and where encephalitis lethargica has been epidemic at one time or another (England and Wales, Sweden, Finland, Denmark, Switzerland and Italy)." In the second column, the figures were obtained by adding together all official reports available for this Survey. For example, reports more or less complete, were obtained for seven countries in 1919; whereas in 1925, seventeen countries are represented.

"Mortality statistics, in the few countries where deaths from encephalitis lethargica are specified, are probably more complete and comparable than the morbidity records. It would appear from such statistics that the disease has been more prevalent in the United Kingdom, Sweden and Switzerland than in other [European] countries. . . . For the period 1921-26, the death-rate (per 100,000 inhabitants) for this disease was 2.5 in England and Wales, 2.5 in Scotland, 2.7

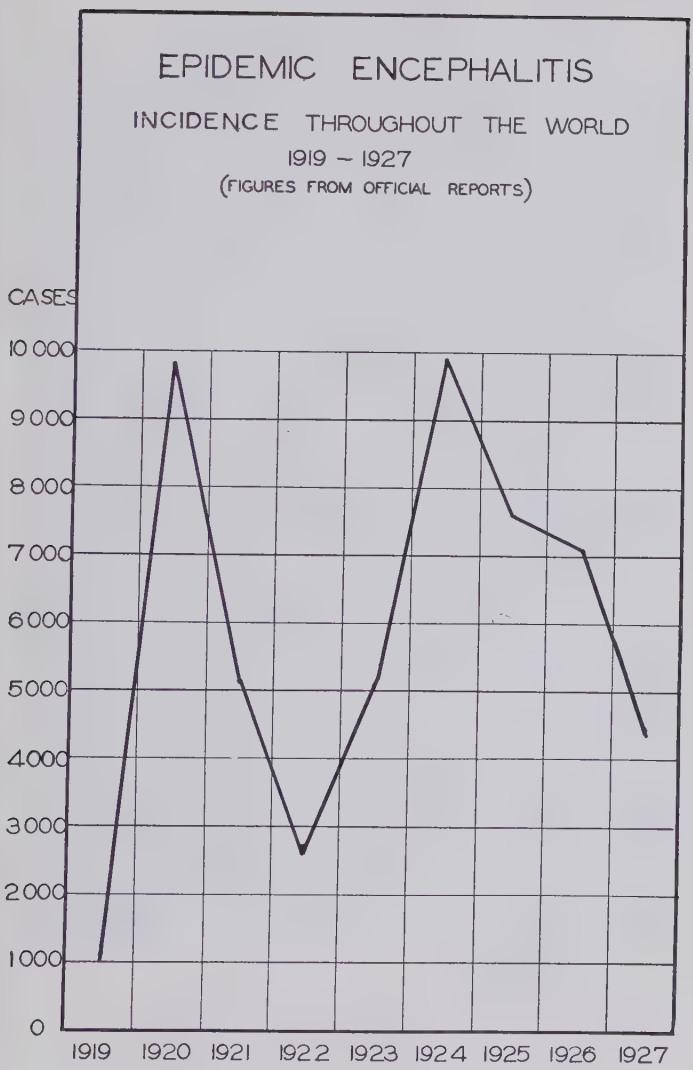
Table Ia

WORLD INCIDENCE OF EPIDEMIC ENCEPHALITIS

	Figures taken from *League of Nations Monthly Epidemiological Reports*, for six European countries	Figures taken from all available official reports (Chart I)
1919	.....	962
1920	7,697	9,824
1921	4,649	5,139
1922	1,273	2,601
1923	2,205	5,244
1924	6,196	9,877
1925	3,766	7,606
1926	2,991	7,141
1927	2,168	4,387
1928 (first half)	1,050	
Total	31,995	52,781

in Northern Ireland, 2.6 in Sweden, 2.8 in Switzerland (1920-25), and 2.6 in Malta. It was somewhat lower in Denmark (1.6) and in the United States registration area for deaths (1.5). Still lower rates were obtaining in Australia (1.0), in New Zealand (0.8) and in the Netherlands (0.8). The highest death-rates for any single year were reported in 1920 in Switzerland (7.5), and in 1921 in Sweden (6.3). In recent years, however, the incidence has been only half as high in Sweden and Switzerland as in the United Kingdom: 1.8 in Sweden and Switzerland and 3.5 in England and Wales (1924-26)."

Chart I


EPIDEMIC ENCEPHALITIS
INCIDENCE THROUGHOUT THE WORLD
1919 – 1927
(FIGURES FROM OFFICIAL REPORTS)
CASES
10 000
9 000
8 000
7 000
6 000
5000
4000
3000
2000
1000
0
1919
1920
1921
1922
1923
1924
1925
1926
1927

Seasonal Incidence

Epidemic encephalitis is a disease of the winter months. This was readily apparent to all observers during the first years of the epidemic and is borne out, with but few exceptions, by the earlier statistical reports. An increase of cases in November and December of one year preceded a sudden rise to a peak during the first quarter of the following year. This is shown in the graph illustrating the incidence in England (Chart IV), in the curve for New York City (Chart V), and in that for 7,000 cases in the literature reports (Charts III and IIIa). In Switzerland 826 of the 984 cases reported for 1920 fell in January, February and March (Charts VI and VIII). More than half of the 177 cases observed by Netter in France from November, 1919, to November, 1920, occurred in December, January and February (Chart VIII). In Poland, however, the peak of the great epidemic of 1920 was in April, May and June (Chart VIII); in Sweden, there was a secondary rise in the summer of 1920 (Chart VII). In England (Chart IV) and in Scotland in 1924, the epidemic was at its height in May, June and July. In fact, the total of available official reports for 1924 shows the peak of the world incidence for that year to be in May. The total for the years 1919-27, however, shows a predominance in February and March.

The "mysterious disease" in Australia and the Japanese epidemic of 1924, on the contrary, occurred in the late summer and early fall. Cleland and Campbell refer to the occurrence of the disease in New South Wales "towards the end of the hot, dry summer season, ... months characterized by relative dryness and a bracing atmosphere with cold and sometimes frosty nights."

Kaneko comments upon the heat and drought of the summer of the great epidemic in Japan. In China, too, although the winter months—"the period of dry cold and dust winds"—showed the greatest incidence, August, the beginning of the dry season, came next.

In Zamboanga, Philippine Islands, the cases reported for 1923 occurred during a season when it was "very hot during the day and very cold at night."

The characteristic preference of the European and American form of epidemic encephalitis for the colder months has, however, undergone a change during the last few years. The *League of Nations Monthly Epidemiological Report* for February, 1927, comments as follows:

"This disease, the early manifestations of which were of a clearly epidemic character has, as a matter of fact, during the last two years shown a distinct tendency towards attaining an endemic level subject to moderate seasonal fluctuations." And again in September, 1927, we have the statement: "Seasonal fluctuations are becoming more and more uncertain; there was thus a slight increase of the number of cases in June in several countries of northern Europe."

The figures given in Table II (Chart II) were obtained from such official reports as were available for this Survey. The yearly totals are as accurate as the original figures from which they are compiled. The figures for the months have only a relative degree of accuracy. In certain cases the original report was given by quarters; in such instances the total was divided equally among the respective months. In other cases, the figures given for the months did not add up to the yearly total; in these instances the difference was divided proportionally among the months.

For 1919, reports were obtained from seven countries; for

Table II (Chart II)

World Incidence of Epidemic Encephalitis by Months [1919-27] as Shown by Available Official Reports

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1919	141	180	189	56	63	17	30	26	27	50	56	127	962
1920	1,187	3,793	1,371	1,156	590	516	304	191	153	103	158	302	9,824
1921	1,359	1,231	566	340	255	204	196	158	209	216	171	234	5,139
1922	184	221	260	198	195	169	132	220	242	242	246	292	2,601
1923	592	1,162	1,204	524	269	231	182	145	214	272	221	228	5,244
1924	383	520	883	1,298	1,625	1,479	1,022	558	472	493	483	661	9,877
1924*	.....	.....	.....	.....	.....	.....	.....	4,985	2,592	895	.....	.....	16,826
1925	682	729	913	697	786	670	602	486	566	530	480	465	7,606
1926	720	753	833	608	603	578	570	500	487	457	491	541	7,141
1927	445	406	498	382	467	340	340	321	271	346	281	290	4,387
Total	5,693	8,995	6,717	5,259	4,853	4,204	3,378	2,605	2,641	2,709	2,587	3,140	52,781

* Figures for the Japanese epidemic included.

Chart II

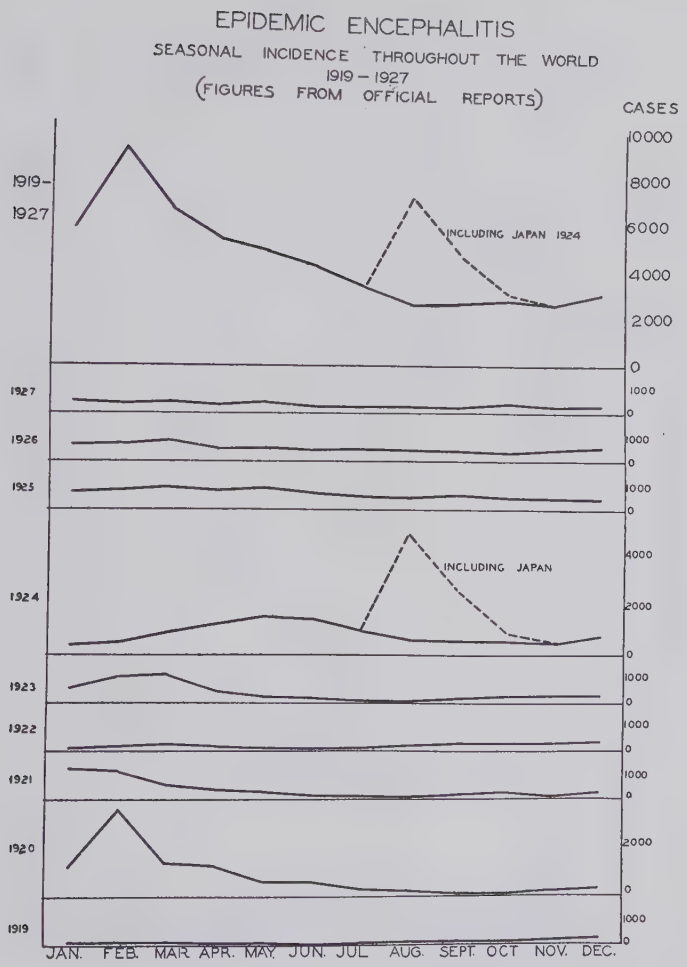

EPIDEMIC ENCEPHALITIS
SEASONAL INCIDENCE THROUGHOUT THE WORLD
1919 – 1927
(FIGURES FROM OFFICIAL REPORTS)
CASES
1919–1927
INCLUDING JAPAN 1924
10000
8000
6000
4000
2000
0
1927
1926
1925
1924
1923
1922
1921
1920
1919
1000
INCLUDING JAPAN
JAN. FEB. MAR APR. MAY JUN. JUL AUG. SEPT. OCT NOV. DEC.

Table III (Charts III and IIIa)

Incidence by Months of 7,876 Cases of Epidemic Encephalitis Reported in the Literature *

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	N.S.	Total
1915	.....	1	1	2	1	1	.....	1	1	.....	.....	2	.....	10
1916	1	1	1	0	1	1	1	1	4	4	4	7	2	28
1917	9	23	21	13	10	4	1	2	2	0	1	5	8	99
1918	19	20	71	50	40	16	9	11	10	30	20	60	102	458
1919	120	163	106	48	28	17	22	37	32	66	128	303	223	1,293
1920	918	707	444	249	178	105	88	50	73	44	71	190	319	3,436
1921	202	110	56	28	17	28	16	21	15	5	22	28	136	684
1922	29	34	35	19	15	11	7	11	6	15	19	57	62	320
1923	104	133	81	16	9	12	16	7	3	5	8	12	32	438
1924†	31	21	34	24	14	14	8	5	7	8	5	16	54	241
1925	10	10	7	8	5	5	5	5	3	5	5	7	17	92
1926	6	7	6	6	2	4	3	5	4	1	3	1	12	61
Total	1,449	1,230	863	463	320	218	176	156‡	160	183	286	688	1,684§	7,876

* Deaths recorded, 3,980 (50.53 percent).

† In addition, 1,579 cases were reported in Japan in August by Iimura and Katsumata.

‡ The August total, including the Japanese cases, is 1,735.

§ Including 716 cases in which the year of onset was not stated.

The marked decrease it the total number of cases reported for the years following 1920 is not to be taken as indicative of the true incidence for these years, but rather of a lessened interest in reporting case histories.

CHART III

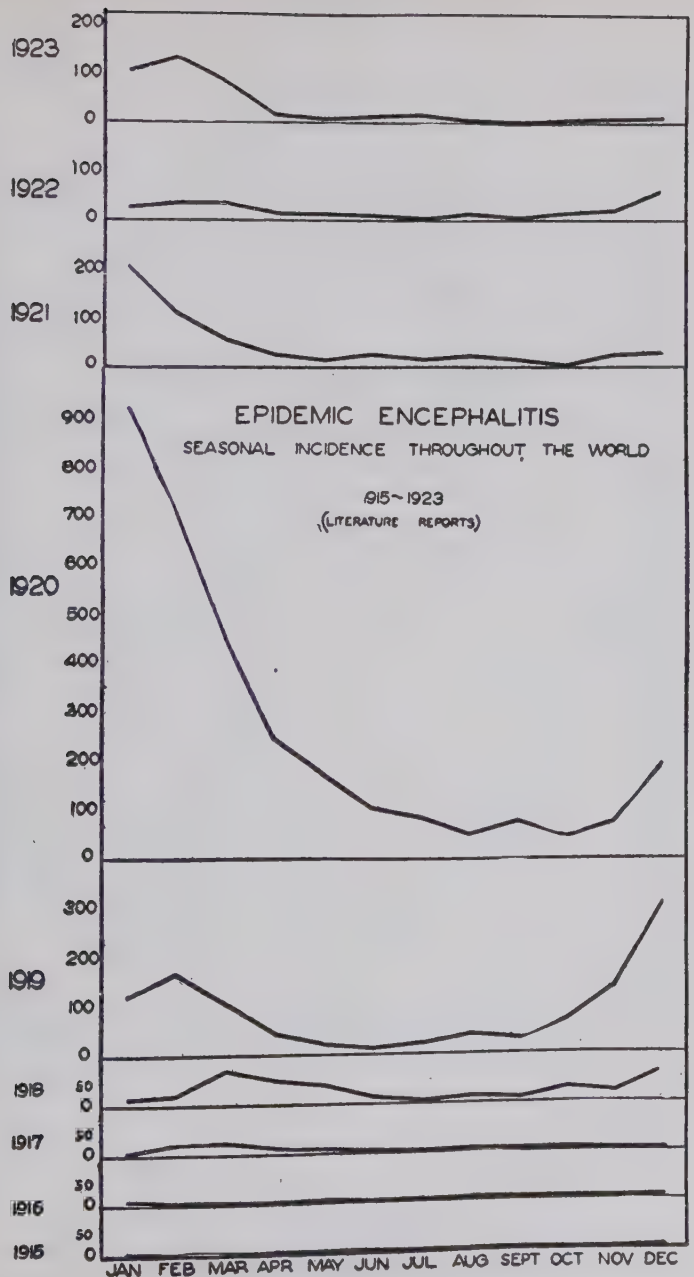

1923
200
100
0
1922
100
0
1921
200
100
0
EPIDEMIC ENCEPHALITIS
SEASONAL INCIDENCE THROUGHOUT THE WORLD
1915~1923
(LITERATURE REPORTS)
1920
900
800
700
600
500
400
300
200
100
0
1919
300
200
100
0
1918
50
0
1917
50
0
1916
50
0
1915
50
0
JAN FEB MAR APR MAY JUN JUL AUG SEPT OCT NOV DEC

CHART IIIa

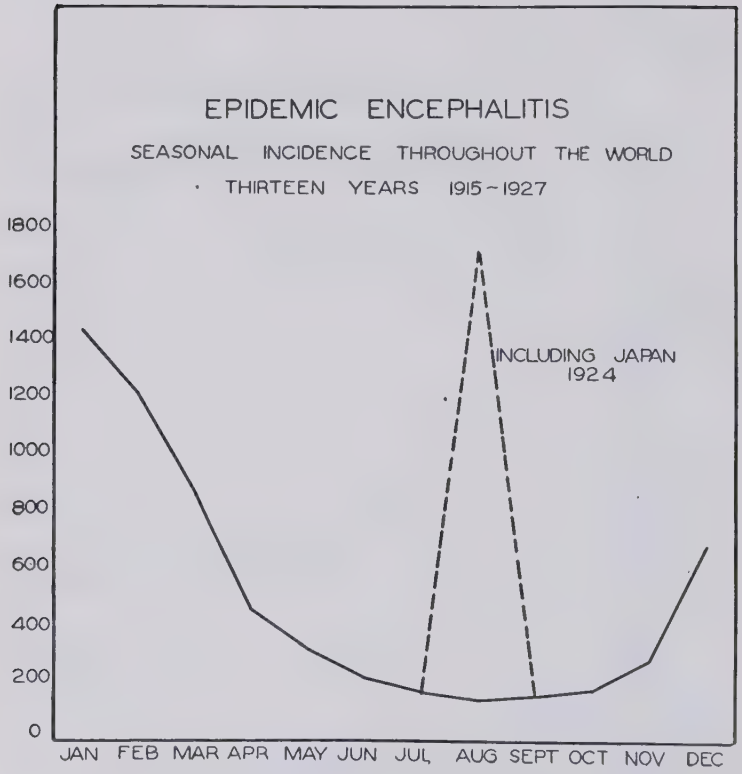

EPIDEMIC ENCEPHALITIS
SEASONAL INCIDENCE THROUGHOUT THE WORLD
THIRTEEN YEARS 1915~1927
INCLUDING JAPAN 1924
1800
1600
1400
1200
1000
800
600
400
200
0
JAN FEB MAR APR MAY JUN JUL AUG SEPT OCT NOV DEC

CHART IV

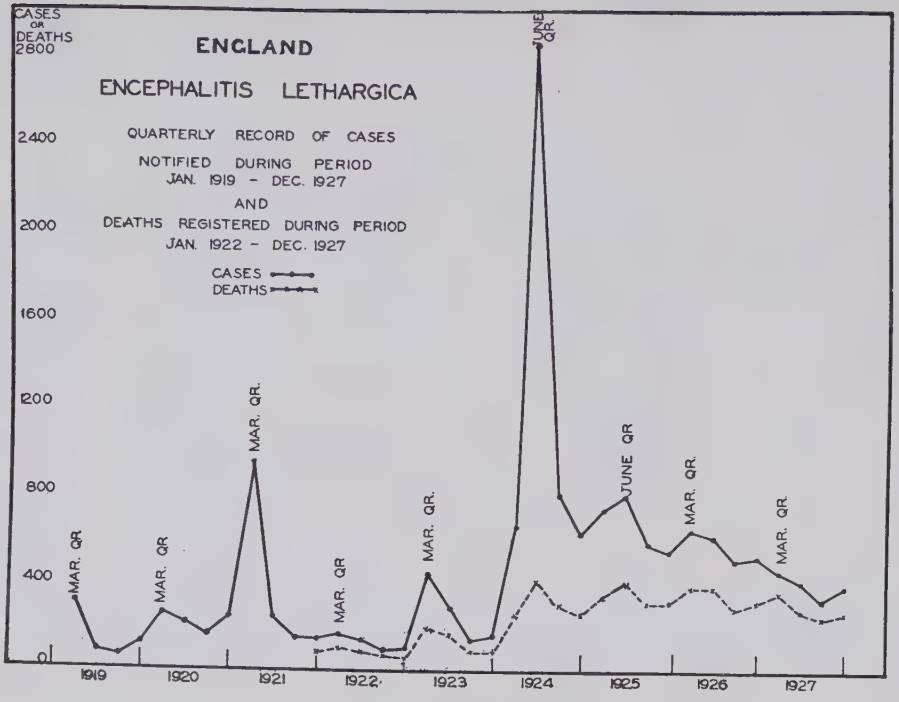

CASES OR DEATHS
2800
2400
2000
1600
1200
800
400
0
ENGLAND
ENCEPHALITIS LETHARGICA
QUARTERLY RECORD OF CASES
NOTIFIED DURING PERIOD
JAN. 1919 - DEC. 1927
AND
DEATHS REGISTERED DURING PERIOD
JAN. 1922 - DEC. 1927
CASES
DEATHS
MAR. QR
MAR. QR
MAR. QR.
MAR. QR
MAR. QR.
JUNE QR.
JUNE QR
MAR. QR.
MAR. QR
1919
1920
1921
1922
1923
1924
1925
1926
1927

CHART V

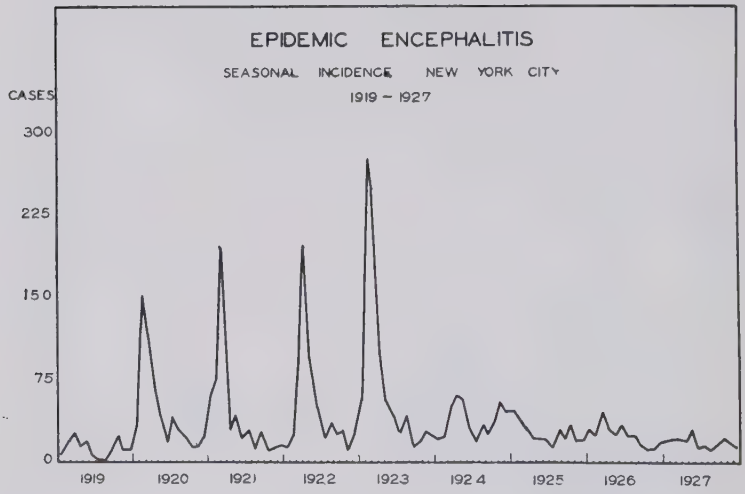

EPIDEMIC ENCEPHALITIS
SEASONAL INCIDENCE NEW YORK CITY
1919 – 1927
CASES
300
225
150
75
0
1919
1920
1921
1922
1923
1924
1925
1926
1927

CHART VI

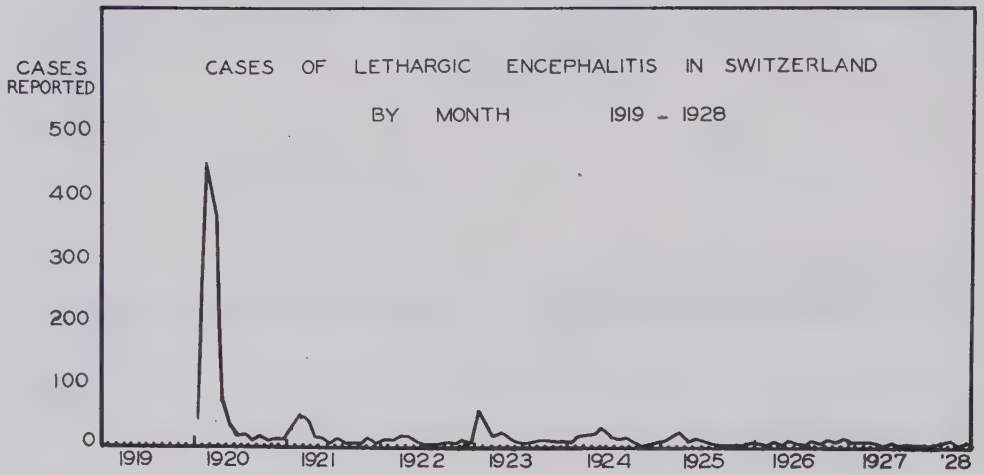

CASES OF LETHARGIC ENCEPHALITIS IN SWITZERLAND
BY MONTH 1919 - 1928
CASES REPORTED
500
400
300
200
100
0
1919
1920
1921
1922
1923
1924
1925
1926
1927
'28

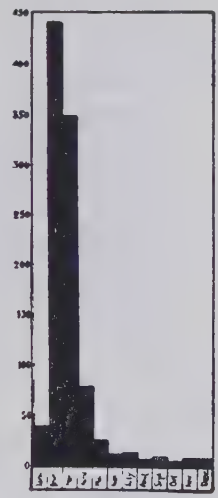
CHART VII

CASES OF LETHARGIC ENCEPHALITIS IN SWEDEN
BY MONTH, 1919 – 1928
CASES REPORTED
500
400
300
200
100
0
1919
1920
1921
1922
1923
1924
1925
1926
1927
1928

Chart VIII

Seasonal Incidence of Epidemic Encephalitis

France, 1920.



Suisse, 1920.

Ville de New-York, 1920.

Pologne, 1920.

Reproduced from Van Boeckel, Bessemans and Nelis, V 19

1920, from ten; for 1921, from eight; for 1922, from ten; for 1923 and 1924, from fifteen; for 1925 and 1926, from seventeen; and for 1927, from sixteen.

Age, Sex, Occupation and Race

Distribution by Age

Epidemic encephalitis may occur at any age. It has been reported in the newborn by several writers, Harris (H 74), Parsons (P 47a), Bompiani (B 277), Jorge (J 61), and Ladroitte (L 14). At the other extreme, Netter is quoted as having seen a patient of eighty-seven years; MacNalty (M 23) refers to the death of a woman of eighty-four years from this disease and Leiner (L 114) speaks of age incidence "to 96 years."

The age period of greatest incidence varies somewhat with the group of cases reported. Thus both in Dr. Neal's series of 274 cases and in the Happ and Mason series of 79, 50 percent were under fifteen years of age. These authors see a larger proportion of children than do many of those who have reported on epidemic encephalitis. It may be that many cases in the younger age periods are overlooked because of the greater difficulties in the diagnosis. The high incidence among children, especially in the age period under five years, was a conspicuous feature of the "mysterious disease" in Australia in 1917-18, and is one argument advanced against the identity of this disease with epidemic encephalitis.

In 1,273 cases in England and Wales in 1919 and 1920, the highest incidence (25.6 percent) was in the age group 10-20 years. Redlich reported 45 out of 160 cases (28.9 percent) in Vienna, and Stiefler 15 out of 59 cases (25.4 percent) in

Chart IX

Distribution by Age, Showing Incidence (Cases) by Age Groups of 50 Cases Reported by Netter and of 59 Cases Reported by Stiefler

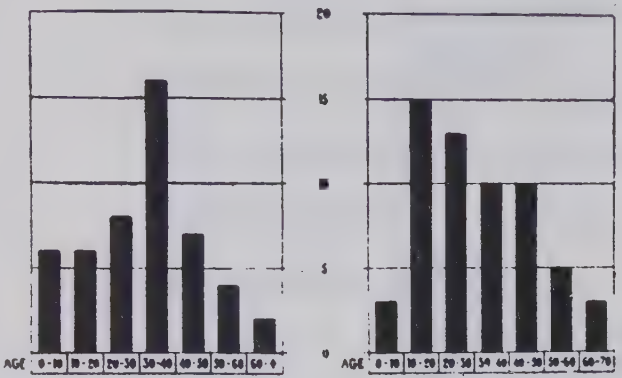


Fig. 48.
Statistique de Netter.
France.

Fig. 49.
Statistique de Stiefler
Autriche.

Reproduced from Van Boeckel, Bessemans and Nelis, V 19

Chart X

Distribution by Age, Showing Incidence (Percent) by Age Groups of 1,374 Cases Reported by Sterling

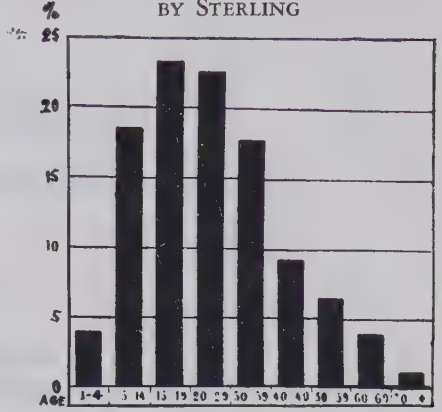


Fig. 50
Statistique de Sterling.
Pologne.

Reproduced from Van Boeckel, Bessemans and Nelis, V 19

Chart XI

Distribution by Age, Showing Incidence (Percent) by Age Groups of 864 Cases Collected by Strauss and Wechsler and 528 Cases Reported by Carrière

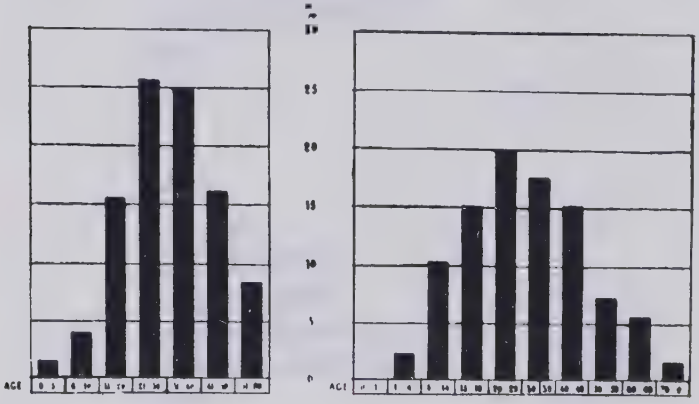
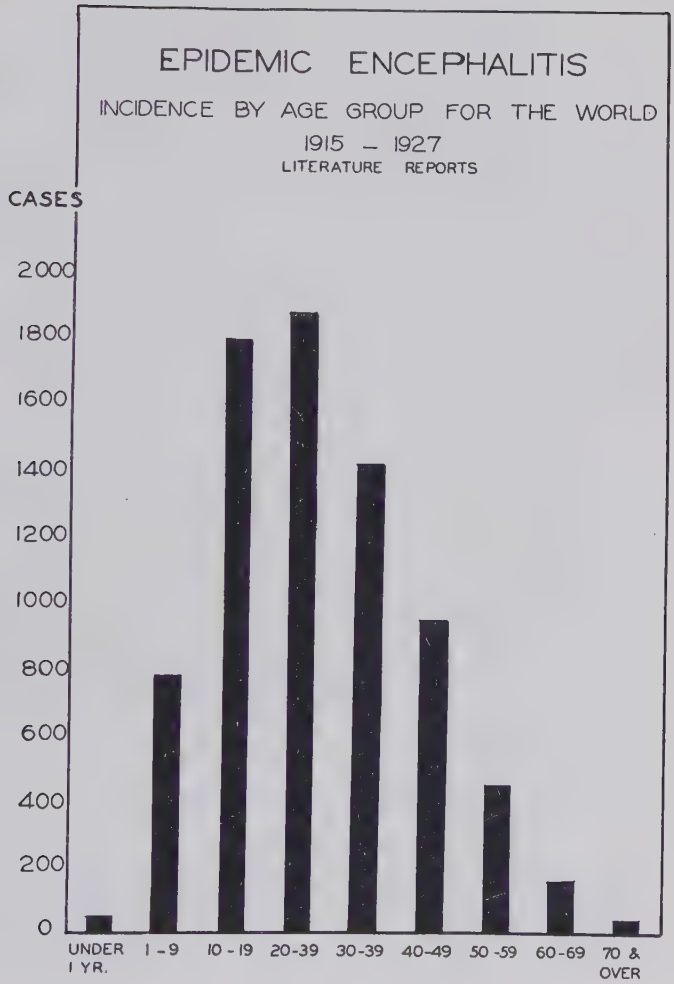


Fig. 47.
Statistique de Strauss et Wechsler. États-Unis d'Amérique.

Fig. 46.
Statistique de Carrière. Suisse.

Reproduced from Van Boeckel, Bessemans and Nelis, V 19

CHART XII


EPIDEMIC ENCEPHALITIS
INCIDENCE BY AGE GROUP FOR THE WORLD
1915 – 1927
LITERATURE REPORTS
CASES
2000
1800
1600
1400
1200
1000
800
600
400
200
0
UNDER 1 YR.
1 – 9
10 – 19
20-39
30-39
40-49
50 -59
60-69
70 & OVER

Linz in the same group. In the Sheffield epidemic the incidence in this group was 30.9 percent. In the Polish outbreak of 1920, Sterling found 315 (22.9 percent) of 1,374 cases in the five-year group 15-19 years, a higher percentage than in any ten-year period for other ages. Fifty-five of 209 cases (26.3 percent) reported by Madsen in Denmark were in persons between 15 and 25 years of age; in New York City, 88 (16.5 percent) out of 531 cases reported in 1926 and 1927, came within this same age period. Strauss and Wechsler's 864 collected cases showed a percentage of 25.7 in the age period 21-30 years. This same age group was also the highest (34 percent) in Carrière's series of 528 cases in Switzerland; and in Hoff's cases in Vienna (38.4 percent). On the other hand, 32 percent of Netter's fifty cases fell within the age group of 30-40 years.

In the Japanese epidemic of 1924, the higher age groups were most affected; out of 2,936 cases, 668 (22.7 percent) and 646 (22.1 percent) were found to be in the ten-year periods 61-70 years, and 71-80 years, respectively.

Of 9,680 cases collected for this Survey from the literature reports of all countries, the age was stated in 7,584 cases. Of these the age group 20-30 years showed the highest incidence; 1,921 cases (25.3 percent) came within this period; the 10-20 year group stands next with 1,802 cases (23.7 percent) (Table IX, Chart XII).

Thus it would appear that, with few exceptions, young adults are most susceptible to epidemic encephalitis.

Distribution by Sex

The majority of statistical reports show a greater incidence among males than among females. The records for the Ministry of Health (Great Britain) for 1919 and 1920,

to be sure, indicate an almost equal division between the sexes: 634 males (49.8 percent) and 639 females (50.2 percent). However, in the Sheffield epidemic in 1924 we find the usual predominance of males. 186 males (59.3 percent) to 131 females (40.7 percent). In Vienna, Hoff found 642 males (57.6 percent) to 471 females (42.4 percent); in Switzerland, Carrière reported 278 males (52.5 percent) to 250 females (47.4 percent). Of 531 cases in New York City in 1926 and 1927, 55.45 percent occured in males. Netter, on the other hand, in a small series of 70 cases found 29 (41.4 percent) were in men and 41 (58.6 percent) in women. Of 8,537 cases collected from the world literature, in which the sex was stated, 4,935 (57.8 percent) were males, and 3,602 (42.2 percent) females.

Van Boeckel, Bessemans and Nelis do not find these figures significant of a specific electivity of encephalitis for the sexes; in general, they say, men are more exposed to infection than women are, and the figures for this disease are not out of proportion to the probable exposure.

There is more variation in the results of analyses based on the relationship of sex incidence to age periods. Parsons found "that the scales are held fairly even all through the age periods" (Table 3, England and Wales). Certain authors note a greater incidence among females in the earlier age groups, with a reversed ratio at later ages. Wynne's figures for the Sheffield epidemic, for example, show more females (11) than males (3) in the period under 5 years. The same is true of Carrière's report; under 5 years, there were 5 males and 7 females. On the other hand, in Goldflam's series there were 7 males and 1 female in the period under 10 years and, in Goldstein's 3 males and no females in the same group. These authors do not report the period "under 5 years" separately.

It will be noted that the total number in the lower age groups in all the above series of cases, is small. In the New York City cases of 1920 we have: under 5 years, 23 males, 24 females, 5 to 9 years, 26 males, 14 females.

Dr. Neal (personal communication) reports 146 cases under 10 years seen in New York City from September, 1918 to June, 1922, as follows:

Table IIIa

Sex Incidence in Cases Under 10 Years

[Neal, New York]

	Age period					
	Under 5 years			5–9 years		
	M	F	Total	M	F	Total
1918	1	2	3	1	2	3
1919	14	4	18	7	4	11
1920	13	11	24	15	5	20
1921	17	12	29	13	9	22
1922	7	5	12	1	3	4
Total	52	34	86	37	23	60
Percentage	60.4	39.6	100	61.6	38.4	100

Thus in this larger series, 60 percent were males in both age groups.

In his review of the epidemic in Glasgow, Chalmers (C 84) gives figures for the ages under discussion (Table IIIb).

Here again we find a high percentage of males in both groups.

Of the cases collected from the world literature, 5,859 have been analysed with regard to age and sex incidence (Table IV,

Chart XIII.) Of this number, 577 were under 10 years; of these 56 percent were males and 40.5 percent females. (Sex not stated, 3.5 percent.)

Table IIIb

SEX INCIDENCE IN CASES UNDER 10 YEARS
[CHALMERS, GLASGOW]

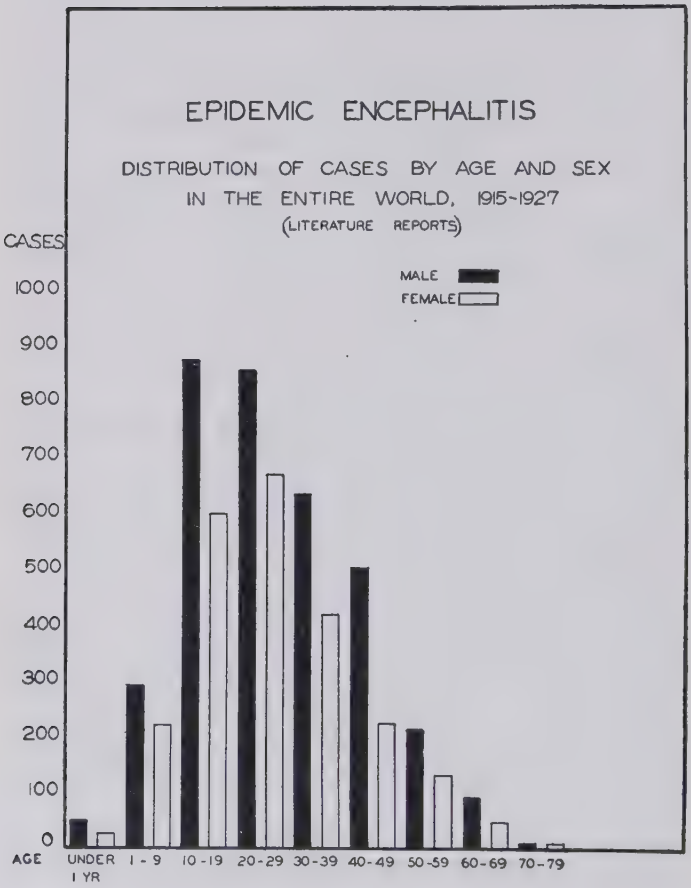
	M	F	Total	Percentage of males
Under 5 years	23	11	34	66
5–10 years	50	28	78	64
	73	39	112	

Table IV

AGE AND SEX INCIDENCE OF 5,859 CASES COLLECTED FROM THE LITERATURE

Age group	Cases			Percentage			Total
	M	F	NS	M	F	NS	
Under 1 year	27	14	3	61	31	8	44
1–9	297	220	16	56	41	3	533
10–19	871	593	24	58	39	3	1,488
20–29	849	668	20	55	44	1	1,537
30–39	627	416	11	59	40	1	1,054
40–49	503	225	6	68	31	1	734
50–59	208	127	5	61	38	1	340
60–69	71	38	.....	65	35	.....	109
70–79	10	10	.....	50	50	.....	20
Total	.....	.....	.....	.....	.....	.....	5,859

CHART XIII


EPIDEMIC ENCEPHALITIS
DISTRIBUTION OF CASES BY AGE AND SEX
IN THE ENTIRE WORLD, 1915-1927
(LITERATURE REPORTS)
CASES
MALE
FEMALE
1000
900
800
700
600
500
400
300
200
100
0
AGE
UNDER 1 YR
1 - 9
10 - 19
20 - 29
30 - 39
40 - 49
50 - 59
60 - 69
70 - 79

A study of this table shows little variation in the sex incidence at the different age periods indicated. Approximately 60 percent of the total number of cases in each ten-year group are males; the lowest percentage of males (55) is found in the 20-29 year group and the highest (68 percent) at 40-49 years. In general, the male incidence seems to be slightly greater above the age of 40 years than below that age.

Occupation and Social Condition

"There is no indication of any connection between occupation and liability to infection," Hall (H 29a). James (J 25) found "among the adult male patients nearly as many occupations as there were cases of illness." Professor Wynne's (W 151) study of the occupations involved and of the social status of the patients in the Sheffield epidemic, led him to conclude that there was no relation between the disease and social conditions; the occupations were affected in due proportion to the population. Strauss and Wechsler (S 384) found "every conceivable occupation represented." Sterling (S 318) also concluded that neither social nor economic conditions played a rôle in the development of the disease; he listed 34 occupations but found no one affected unduly.

Parsons (P 47a) (Table 6, England and Wales), however, attempted a classification in the case of 1,070 patients for whom information was available and concluded "that the disease attacks chiefly those individuals who spend the greater part of the day indoors." On the other hand, d'Arbela and Montanari (D 31a) found the greatest number of their cases among the working and peasant classes. Kaneko and Aoki (K 14a) also found the incidence greatest among the peasants or farmers, and attributed it to overwork and exposure

to the hot sun while watering their rice fields during the drought. A study of the cases in Vienna led Hoff (H 189) to conclude that members of the professions and skilled laborers were noticeably affected, the metal workers particularly. In spite of the prominence of the metal industry in Austria, Hoff feels that the number of patients from this trade is relatively high.

So varied are the occupations recorded in the 7,000 cases collected from the literature for this Survey, that no attempt has been made to classify them. It seems fair to conclude that neither occupation nor social condition bears any relationship to the susceptibility to epidemic encephalitis.

Race

Very little comment has been made in the literature on racial incidence. In Warsaw, Sterling found that the proportion of the Jewish race affected was high, compared with the population. No other reports confirming this observation have been found. Of the cases seen in India, South Africa and the Philippines, a high percentage occurred among the natives. The mortality too was high. These groups were all small, however. As no large epidemics have occurred in any of these localities, no conclusion could be ventured on the basis of these reports. In the United States the negro has not proved to be especially susceptible to the disease, at least if one may judge from the mortality statistics. According to the figures for mortality from encephalitis lethargica in the registration area of the United States, 1921-25 (Tables VI and VII), the incidence of mortality is higher among the white than among the colored population (*League of Nations Monthly Epidemiological Reports*). Except for the great outbreak in Japan

in 1924, there has been nothing to suggest an unusual susceptibility of the yellow race to this disease.

The incidence of epidemic encephalitis has undoubtedly been greatest among the nations of Europe and in the United States. Whether this is due to a greater susceptibility of the white race or to the conditions, as yet undertermined, that were responsible for the recent epidemic, is a question that cannot yet be answered.

Mortality

The figures with respect to mortality vary greatly with the groups of cases reported. The low case mortality percentage (7.4) in Happ and Mason's series of 81 cases is unusual, especially in view of the fact that so many of their patients belonged to the lower age groups, where the mortality rate is frequently high. Goldflam, of Warsaw, reported only 11.3 percent mortality in his group of 126 cases. Sterling's figures from Warsaw were also low (19.3 percent.) Hall refers to the mortality (15.5 percent) of von Economo's early group of 13 cases and believes that "these figures probably represent the general mortality very fairly." Most writers, however, give a higher percentage. Of Cruchet's 48 early cases at Bar-le-Duc, 37.5 percent were fatal. Chalier, of France, (C 81a) places the mortality at from 40 to 50 percent; Madsen, of Denmark, at 46 percent; Parsons, of England, at 48 percent; Smith, of the Philadelphia General Hospital, 41 percent; McClure, of Manchester, England, (M 203) 53 percent; and Butt, of South Africa, 92.3 percent (13 cases).

Intermediate figures appear more frequently: Bouman, of Holland, reports a mortality of 27.5 percent; Van Boeckel, Bessemans and Nelis, of Belgium, 31 percent; Boyd, of Can-

ada, 39 percent in the first Winnipeg epidemic, and 25 percent in the second; Chalmers, of Scotland, 20 percent; James, of England, 20 percent; Almasio, of Italy, 34 percent; d'Arbela and Montanari, of Italy, 27 percent; Gottstein, of Prussia 35.6 percent; Carrière, of Switzerland, 29.4 percent; Pappenheim, of Russia, from 14.3 to 25 percent; Dunn and Heagey, of the United States, 31 percent; Strauss and Wechsler, also of the United States, 20 percent; N. R. Smith, of Michigan, (S 246) percent; and Price, of Spokane, Washington, 24 percent. Hall finds that "on the whole the average works out at about 27 percent."

Of 7,876 cases collected from the literature for this Survey, a fatal result was recorded in 3,980 instances (50.53 percent.)

A considerable variation is also shown in official reports. The case fatality rate in Prussia as computed by Deicher for

Table IVa

Case Fatality in Epidemic Encephalitis

Year	England and Wales	New York City
	Percentage	Percentage
1919	54.3	29
1920	53.9	45
1921	49.6	37
1922	74.6	33
1923	51.8	38
1924	27.9	53
1925	52.1	57
1926	58.5	62
1927	71.6	69

the years 1919 to 1924 was 14.9 percent, but this figure is exceptionally low. Even in the same country the mortality rate has fluctuated with the epidemics of different years. The figures for England and Wales and those for New York City for the years 1919-27 are given below for comparison.

A discussion of this subject is given in the *League of Nations Monthly Epidemiological Report,* R. E. 116, July 15, 1928. Portions of this article are quoted here:

"The case mortality rate of encephalitis lethargica is variable and, owing to inaccuracy in notification, difficult to determine. Based upon the 15,935 cases officially reported and the 7,632 deaths registered in England and Wales from 1919 to 1927, it was 47.8 per cent. This figure, however, is undoubtedly too high, as proved by special investigation; in a series of 3,558 traced and investigated cases, the rate worked out at 33.5 per cent, and has varied considerably from year to year. During the extensive epidemic of 1924, it was 27.9 per cent, while during the other years it has averaged 57.1 per cent. These figures do not differ greatly from those reported in other countries, where the notification may be assumed to be fairly good. In Switzerland, during the epidemic of 1920, the case mortality rate was 29.5 per cent; during the years 1921 to 1925 it was 64.2 per cent. In Sweden the case mortality rate was 27.0 per cent during the years 1920 and 1921, when the disease was epidemic; it was 37.0 per cent during the years 1922 to 1924 and 57.5 per cent in 1925-26.

"Thus, the case mortality rate was about the same during the large epidemics in Switzerland in 1920, in Sweden in 1920-21, and in England in 1924. The case mortality rate averaged, during the years 1921-26, 54.4 per cent in Denmark; during the years 1921-27, 59.5 per cent in the Netherlands, and, during the years 1921-27, 33.8 per cent in New Zealand,

in none of which countries have there been extensive epidemics."

The question of mortality as affected by age and sex may be considered here. The recently published report of the British Ministry of Health (P 46) is in agreement with Parsons' earlier figures, and shows that the case mortality rate is highest "among those patients who are attacked at the two extremes of life, and that it tends to rise progressively after the age of about twenty-five."

An analysis of the ages at death of 4,419 patients (Table VII), whose deaths were reported during the years 1921-25, showed that "the highest number of deaths appear in the age group 1 to 5 and more deaths fall within the periods 1 to 5 and 6 to 10 than within any other two periods; a comparatively large number of deaths fall also within the age period 36 to 55."

From certain groups of cases it would appear that the relative susceptibility of the sexes has a definite relationship to the age group. Professor Wynne analysed the forty-five deaths in the Sheffield epidemic (Table 8, England and Wales) and found that while "the case mortality generally is slightly higher among females than males there were, nevertheless, ten deaths among males between the ages of fifteen and thirty against only one among the females at those ages, a fact that may have some significance. If confirmed by larger statistics it suggests a greater susceptibility to infection in the central nervous system of the male as compared with the female during the period when sexual maturity is achieved and most active."

Other groups do not bear out the statement that "case mortality generally is slightly higher among females than males." Charts XIV and XV show morbidity and mortality by age

CHART XIV

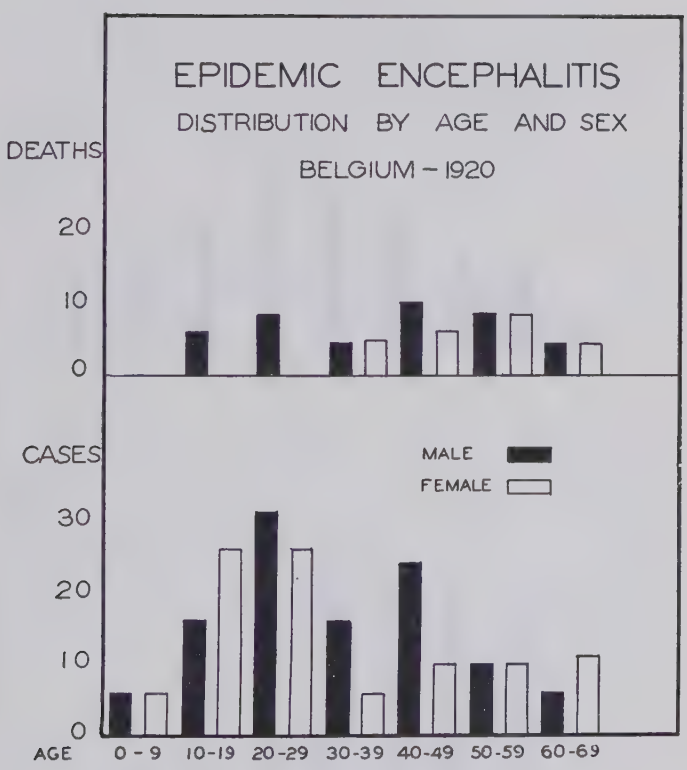
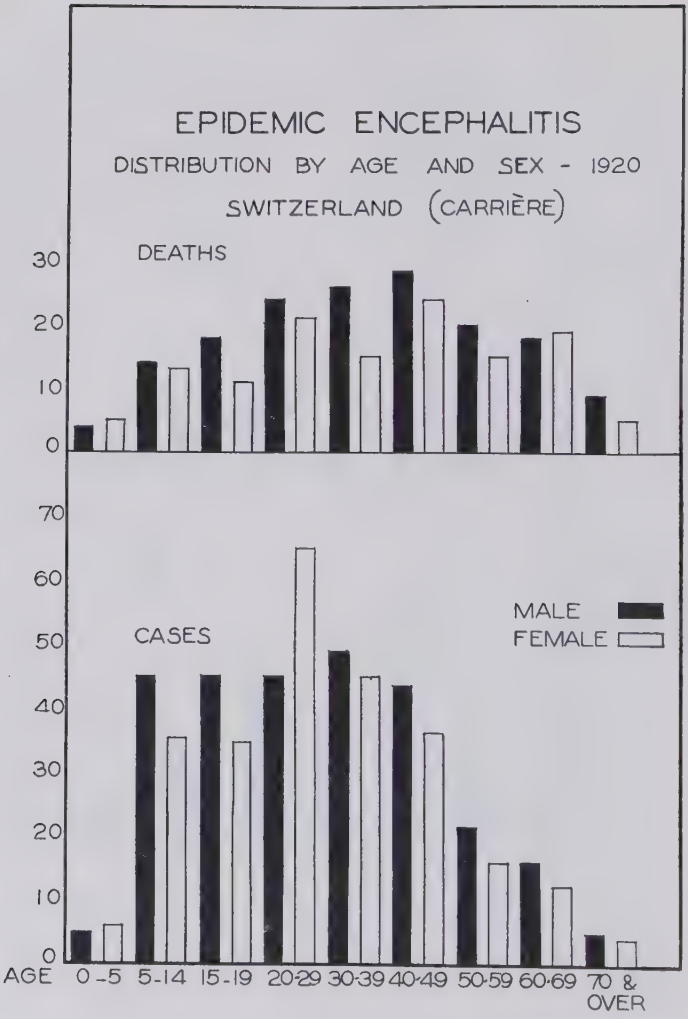

EPIDEMIC ENCEPHALITIS
DISTRIBUTION BY AGE AND SEX
BELGIUM – 1920
DEATHS
20
10
0
CASES
30
20
10
0
MALE
FEMALE
AGE
0 - 9
10-19
20-29
30-39
40-49
50-59
60-69

CHART XV


EPIDEMIC ENCEPHALITIS
DISTRIBUTION BY AGE AND SEX - 1920
SWITZERLAND (CARRIÈRE)
DEATHS
30
20
10
0
70
60
50
40
30
20
10
0
CASES
MALE
FEMALE
AGE
0-5
5-14
15-19
20-29
30-39
40-49
50-59
60-69
70 & OVER

and sex for 200 Belgian cases (Table 2, Belgium), and for 984 cases in Switzerland (Tables 2 and 3, Switzerland).

Pearl (P 62) has analysed by age and sex the New York City report of encephalitis cases and deaths, 1920. (Table 10, United States.) Portions of his conclusion are quoted here:

"*Influence of sex* . . . . it appears that there is a significantly larger proportion of males among those attacked by epidemic encephalitis than there is among the general population. . . . the proportion of males dying from epidemic encephalitis in New York in 1920 was not significantly different from that found in the general population. . . . .

"It thus appears that more males were attacked but no more died from encephalitis than would have happened if sex had no differential effect whatever relative to this disease. This result can only mean that the case fatality rate is different in the two sexes.

"*Influence of age* . . . . it can be asserted, on the basis of the experience of New York City in 1920 with epidemic encephalitis, that in males the disease is probably not significantly more likely to attack one age than another, but instead falls upon males in about the proportion that they are normally present in the general population. It is furthermore not more fatal among the attacked at one age than another until after age 35 is passed. From that point on there are more fatal cases than would be expected if the toll of death were taken purely at random in respect of age . . . . .

"It is quite certain that, so far as the experience of New York City in 1920 may be taken as typical, epidemic encephalitis occurs among females without definite age preference, striking the different ages in due proportion as they are represented in the general population. Females of any par-

ticular age are not more likely to contract the disease than females of any other age.

"The age distribution of deaths among females presents a different case. . . .the death distribution as a whole is significantly divergent from what would happen if the disease was as likely to be fatal at any given age as at any other. . . . . The deaths in the former [age group 5-9] were fewer than expected and more than expected in the latter [age group 35-44]. The numbers are small, however, and it will probably be found, when more data are available, that the facts as to age distribution of deaths are the same in females as in males, namely that there is a definite tendency towards greater fatality in the higher age groups."

From the "Study of Encephalitis Lethargica" in the above-mentioned *League of Nations Monthly Epidemiological Report,* Tables V to VIII and Charts XVI to XXI have been taken, showing mortality by age and sex for a period of years in the countries indicated. The following comment is quoted from this text:

"There has been little difference in the death-rate between the sexes in England and Wales and in the United States; only during the epidemic of 1924 was the death-rate slightly higher for males than for females. During the severe epidemic in Switzerland and in Sweden in 1920-21, however, there was a marked excess of male deaths; in Switzerland, in 1920-21, there were 200 male deaths and 169 female deaths, while during the years 1922 to 1925 there were 134 male and 159 female deaths. In Sweden in 1920-21, there were 283 deaths among males and 221 among females. In the following years, 1922 to 1924, this difference disappeared and there were 193 deaths among males and 186 among females attributed to encephalitis lethargica."

"The mortality rate of encephalitis lethargica is usually highest at the ages between 50 and 60 years. It is also frequently high during the first years of life, but usually decreases very soon. It falls rapidly after 60 years of age. In England and Wales there is little difference in the mortality of the two sexes under 45 years of age. After that time the male mortality is considerably in excess. At the ages between 20 and 24 the female is higher than the male mortality.

"Statistics for Sweden and Switzerland also show that the mortality among males is much in excess after the age of 40 or 50, while before those ages the difference between the two sexes is inconsiderable. In the United States the difference in mortality between the two sexes is inconsiderable throughout life. The incidence of mortality is higher among the white than the coloured population."

Communicability

"The rarity of any evidence of contagion has been noted in every country in which the disease has occurred," (Hall.) In spite of the mass of material (1,156 cases) studied at the Neurological Clinic in Vienna, Hoff (H 189) states that no case of definite contact infection could be established. Not one case of transmission could be traced in the 520 cases reported by Fassbender (F 14), from Germany. Among 11,330 cases in Prussia from 1919 to 1924, Deicher (D 63) found only eight instances of possible contact infection, these involving twenty-seven people. In Japan, Iimura reported 671 cases in 671 families. In Holland, no case of contagion was reported in response to Bouman's questionnaire.

Some writers find convincing evidence of communicability, although the number of cases involved is small. Pecori (P 65)

Table V

MORTALITY FROM ENCEPHALITIS LETHARGICA BY SEX AND AGE IN SWEDEN, [1920-24], AND IN SWITZERLAND [1920-25]

Sweden

Age	Male		Female	
	Deaths	Rate per 100,000	Deaths	Rate per 100,000
0–4	31	2.1	35	2.5
5–14	62	2.1	57	2.0
15–19	56	3.9	51	3.7
20–29	61	2.5	66	2.7
30–39	63	3.3	55	2.7
40–49	76	4.8	51	3.1
50–59	65	5.3	51	3.7
60–69	48	5.1	31	2.8
70+	14	2.0	10	1.1
Total	476	3.24	407	2.67

Switzerland

Age	Male		Female	
	Deaths	Rate per 100,000	Deaths	Rate per 100,000
0–4	18	1.8	17	1.7
5–14	32	1.4	33	1.5
15–19	29	2.5	31	2.6
20–29	50	2.7	59	2.8
30–39	40	2.6	40	2.3
40–49	55	3.8	60	3.9
50–59	60	5.8	41	3.5
60–69	35	5.6	34	4.5
70+	15	4.6	13	2.8
Total	334	2.96	328	2.70

From the *League of Nations Monthly Epidemiological Reports.*

Table VI

MORTALITY FROM ENCEPHALITIS LETHARGICA BY SEX AND AGE FOR THE WHITE RACE IN THE REGISTRATION AREA OF THE UNITED STATES [1921-25]

Age	Male		Female	
	Deaths	Rate per 100,000	Deaths	Rate per 100,000
0–4	487	2.0	495	2.1
5–9	226	1.0	226	1.0
10–14	156	0.7	183	0.9
15–19	226	1.2	246	1.3
20–24	227	1.2	243	1.3
25–29	249	1.3	230	1.2
30–34	274	1.6	243	1.5
35–39	301	1.8	240	1.6
40–44	267	1.9	254	2.0
45–49	288	2.3	265	2.4
50–54	293	2.8	262	2.8
55–59	240	3.0	190	2.6
60–64	168	2.5	165	2.8
65–69	110	2.4	97	2.3
70+	108	1.8	134	2.2
Total	3,620	1.6	3,473	1.6

From the *League of Nations Monthly Epidemiological Reports.*

Table VII

MORTALITY FROM ENCEPHALITIS LETHARGICA BY SEX AND AGE FOR THE COLOURED RACE IN THE REGISTRATION AREA OF THE UNITED STATES, [1921-25]

Age	Male		Female	
	Deaths	Rate per 100,000	Deaths	Rate per 100,000
0–4	29	1.3	37	1.5
5–9	13	0.5	7	0.3
10–14	13	0.5	11	0.4
15–19	27	1.3	18	0.8
20–24	15	0.8	28	1.2
25–29	22	1.3	20	1.0
30–34	19	1.4	15	1.0
35–39	24	1.6	13	0.8
40–44	24	2.0	21	1.8
45–49	11	0.8	10	1.1
50–54	17	1.9	9	1.3
55–59	7	1.3	7	1.8
60–64	3	0.7	2	0.6
65–69	3	1.0	0	0.–
70+	2	0.5	2	0.5
Total	229	1.1	200	0.9

From the *League of Nations Monthly Epidemiological Reports.*

Table VIII

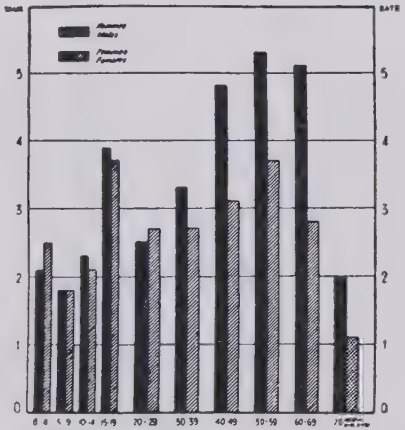
Deaths from Encephalitis Lethargica in England and Wales, by Age and Sex, During the Years 1921 to 1925 Inclusive

Age Groups	Males		Females	
	Deaths	Rate per 100,000	Deaths	Rate per 100,000
0–4	316	3.6	290	3.4
5–9	128	1.6	143	1.8
10–14	190	2.1	155	1.7
15–19	224	2.5	194	2.2
20–24	104	1.3	170	2.0
25–29	99	1.5	126	1.5
30–34	100	1.5	114	1.5
35–39	140	2.2	166	2.3
40–44	148	2.4	165	2.3
45–49	191	3.3	178	2.8
50–54	181	3.5	169	3.1
55–59	167	4.1	126	2.8
60–64	121	3.8	102	2.8
65–69	66	2.9	70	2.5
70+	35	1.2	41	1.0
Total	2,210	2.4	2,209	2.2

From the *League of Nations Monthly Epidemiological Reports.*

Chart XVI

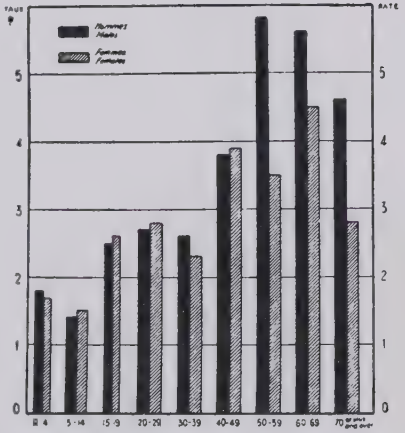
Deaths from Encephalitis Lethargica by Age and Sex in Sweden in 1924.



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928.

Chart XVII

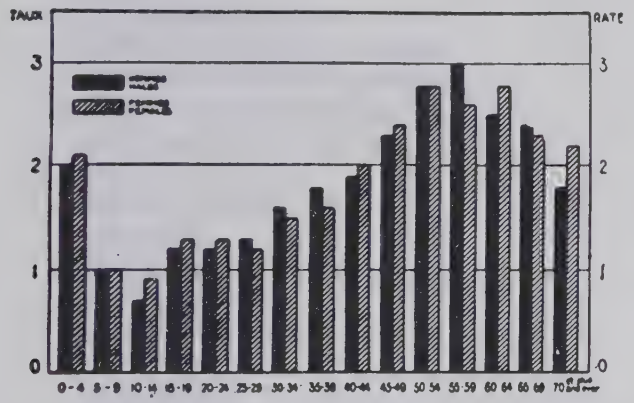
Deaths from Encephalitis Lethargica by Age and Sex in Switzerland, 1920-25.



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928.

Chart XVIII

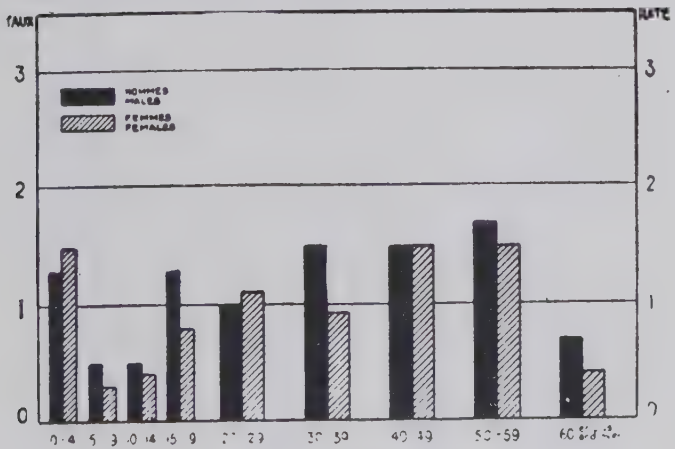
Deaths from Encephalitis Lethargica by Age and Sex Among the White Population of the United States Registration Area, 1921-25



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928.

Chart XIX

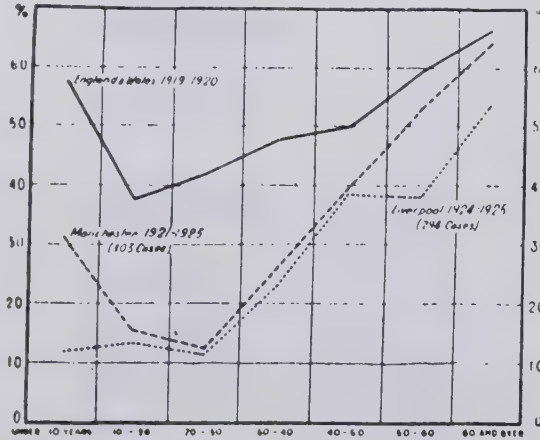
Deaths from Encephalitis Lethargica by Age and Sex Among the Coloured Population of the United States Registration Area, 1921-25



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928.

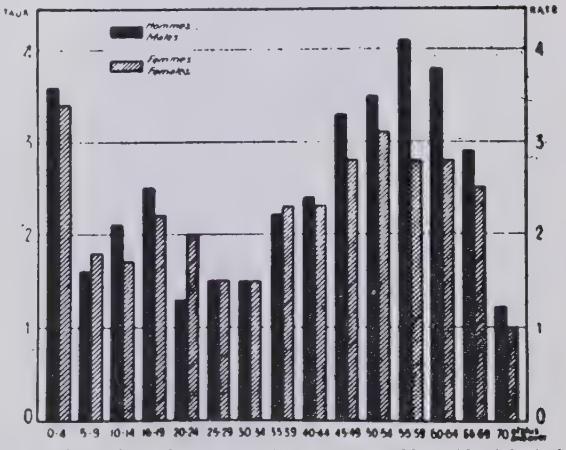
Chart XX

Case Mortality Rate of Encephalitis Lethargica by Age During Various Outbreaks in England



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928

Deaths from Encephalitis Lethargica by Age and Sex in England and Wales [1921-25]



Reproduced from the *League of Nations Monthly Epidemiological Report*, R. E. 116, July 15, 1928

gives eight instances of possible contagion out of 338 cases in Rome in 1919-20, and sets the incubation period from four to fifteen days. Carrière (C 48) gives no specific instances among the 984 notifications in Switzerland in 1920, but says "some familial or house epidemics were observed which leave no doubt of the contagious nature of the disease." Netter (N 55, N 56) determined, he believed, eight clear cases of contagion out of 174 cases studied (4.6 percent). In the Sheffield epidemic Professor Wynne (W 151) also found "evidence of contact, more or less intimate, in six instances involving 13 patients, or just over 4 per cent. of the total number of cases ascertained."

Stiefler (S 357) has described in detail eight instances of probable transmission of encephalitis lethargica from man to man; four of these gave evidence of direct transmission, another was of two abortive cases, in the same building; and three instances were of indirect contact through a supposedly healthy carrier. From a study of these cases, the incubation period was set at from eight to twelve days.

In Liverpool, Stallybrass (S 303) found that direct contact could be ascertained in seventeen out of 106 cases as follows: in the same family twice, involving four persons; contacts with one of these families, two persons; next-door neighbors, three times, seven persons; cousins, once, two persons; and in the same class at school, and neighbors, two persons. Thus the probable source of infection was determined, according to this author, in 9 percent of the cases.

Guillain and Lechelle (G 190) have described the case of a young doctor and his father, which suggests the possibility of transmission through clothing. The father died of epidemic encephalitis in February, 1920. The son journeyed from Paris to the village where his father had lived, occupied his father's

room, sorted his clothing and used some of his accessories, such as handkerchiefs. This was during the latter part of August. In September the son developed epidemic encephalitis.

The evidence in favor of communicability may be considered under the following headings:

Familial or household infections.—Among the more conspicuous instances of this type of infection may be mentioned Kling and Liljenquist's (K 81) study of the epidemic in Lappland in 1921. In this isolated community, all the inhabitants in four small towns were examined. There were many light cases, some atypical, or *formes frustes.* In some families, several members contracted the disease simultaneously; in two households, almost all the members were affected. From their data, the authors estimated an incubation period of ten days.

Van Boeckel, Bessemans and Nelis (V 19) record observations on ten families, forty of fifty-three members affected; four of these families at Ruddervoorde were closely associated and comprised twenty-six persons, seventeen of whom were taken sick; in addition, there were five households with a personnel of thirty-three individuals, eleven of whom developed encephalitis.

Stallybrass (S 304) has also reported multiple abortive cases (twelve) occurring in four household groups.

Parsons (P 47a) found only sixteen instances of multiple infection in families among the 1,293 cases considered in his report on England and Wales. Among Strauss and Wechsler's (S 384) 864 collected cases there were only five instances in which the condition was observed in two members of one family.

Instances in which the disease has been noted in more than one member of a family have been described by many writers

in each country from which literature reports have been obtained. In some instances, as in the case in Mansfield, England, described by MacNalty (M 23), there was a close sequence between the cases; here the first, a child of twenty-one months, was taken sick April 19, 1918; her brother, nine years of age, on May 4; and another brother, seven years of age, on May 8. In other reports an interval of months elapsed between the development of the cases, as in two of the instances described by Netter (N 56); in one family three children were taken sick in January, May and June, respectively; in another family the first case occurred in January, 1919, and the second in June, 1920.

Outbreaks in schools, asylums, or other groups.—The best-known instance of multiple infection in an institution is the explosive outbreak in a girls' home in Derby, England. During August, 1919, twelve cases, with five deaths, developed among the inmates in less than two weeks. A detailed account of this series of cases has been given by MacNalty (M 16). "The source of the outbreak could not be ascertained. The dates of onset appear to indicate that the first two cases derived their infection from a common source. . . . It seems to be uncertain whether personal infection was responsible for the subsequent cases (in which event the incubation period would be of short duration) or whether these cases have derived their infection from the same common source as the first class."

A strikingly similar outbreak occurred in an asylum for young women in Mülheim on the lower Rhine in the summer of 1922. Twenty-eight cases with thirteen deaths occurred in twenty days. This epidemic outburst has been reported, independently, it seems, by John and Stockebrand (J 48a) and Krause (K 127a). Dysphagia, diplopia, and accommodation

paralysis were frequent symptoms. Food-poisoning was at first suspected. The authors evidently remained somewhat in doubt concerning the diagnosis, but Hall and MacNalty report the occurrence together with other outbreaks of epidemic encephalitis.

Fyfe (F 170) saw four cases among the pupils of the same class room in a school in the village of Radway, England. The first case developed on September 15, 1922, the others on October 9, 11 and 13 respectively. All were of the mild ambulatory type.

Epidemics in armies have been rare, but Kristensen (K 146) describes a series of twenty-three cases in the same garrison of the Royal Upland Infantry (Sweden). The first case occurred November 27, 1920, and the last March 7, 1921. The author believes that the infection was spread from man to man directly and estimates the incubation period at from eight to eighteen days.

Cases occurring in hospitals or among doctors and nurses.—Although few in number, several well defined instances of probable contact infection in hospitals wards have been reported. Claude and de Laulerie (C 146) describe one such case. The patient (Case 1) had been in the hospital for Pott's disease since August, 1920. Five cases of encephalitis had been on this service. By September 16, four had been discharged. The remaining patient was suffering from a post-encephalitic syndrome, but was ambulatory and visited among the other patients in the ward. Case 1 developed epidemic encephalitis in December. The day preceding the onset of his illness, a new case of encephalitis had been admitted to the ward.

Guillain, Alajouanine and Celice (G 178) described a similar instance in a patient under treatment for a contracture fol-

lowing poliomyelitis. He had been in the hospital for several years and occupied a bed between two cases of post-encephalitic Parkinsonism. There were no cases of acute encephalitis on the ward at the time. The patient mentioned developed a condition of acute lethargic encephalitis in March, 1924.

The only report of a ward epidemic found is that given by Duzár and Baló (D 211.) These authors describe a rapidly spreading infection which occurred on an infant's ward in Budapest in the fall of 1921. Of seventeen infants, eleven were affected. The incubation period appeared to be from four to six days. Section was obtained on seven cases. The diagnosis of epidemic encephalitis was made on the histological examination of the first case (of fourteen days duration). The other six autopsied cases showed a marked hemorrhagic condition, but not the typical perivascular infiltration; this was attributed by the authors to the fact that in these cases the illness was of briefer duration (two to twelve days.)

Concerning cases developing in hospitals where there are patients with encephalitis, Hall makes the following comment: " . . . . considering the large number of cases of encephalitis lethargica admitted to hospitals in all parts of the world, the fact that such instances are thought worthy of record is the strongest testimony to their infrequency."

More numerous are the reports of instances in which a nurse caring for a patient with epidemic encephalitis has herself contracted the disease. Reece (R 43) has described such an occurrence among the early cases in England, and Lortat-Jacob (L 234) observed a similar case in France in 1920. Among Stiefler's instances (S 366) of possible transmission from person to person, was one where the nurse in a room with six cases of encehalitis also developed a typical case of encephalitis lethargica. Schönfeld (S 111) describes cases of

possible transmission in an insane asylum; a nurse who was caring for a patient with encephalitis lethargica took sick fourteen days later; a patient in the same compartment with a fatal case of encephalitis was taken sick six days later.

Godfrey and Gwyn (G 85) estimate the incubation period at five days, as a result of their observation that a nurse contracted the disease after she had been nursing a patient with encephalitis lethargica for that length of time. Doyle (D 162) also saw a case of epidemic encephalitis in a nurse who had cared for a patient with the same disease, but the time interval is not given.

There were two possible opportunities for infection in the circumstances described by Dewes (D 115.) On January 21, 1920, a physician was present at an autopsy on a case of encephalitis, and while there was stung on the neck by an insect like a fly. Six days later he examined a patient with encephalitis and performed a lumbar puncture. Some of the fluid dropped on his finger. On February 13, he developed encephalitis.

The relatively few recorded instances of infection among doctors and nurses serves to emphasize Parsons' statement "that those in professional attendance upon patients suffering from encephalitis lethargica have very rarely been attacked."

Contact with chronic cases.—Lemierre (L 123) gives the history of a case where the daughter suffered from the after-effects of an acute attack of encephalitis contracted in February, 1918. The father developed the disease in December, 1920, shortly after the daughter had shown a definite reappearance of some of the encephalitic symptoms.

A similar instance was observed by Hall (H 29a.) The possibility of infection during the recurrence of acute symptoms is also suggested by Souques' (S 266) case of conjugal paraly-

sis agitans and Netter's (N 61) case of a child apparently infected by a playmate.

Two instances of possible transmission during an acute relapse of chronic cases are also described by Stiefler (S 357); in these cases there was indirect contact by means of a third person, a clinically healthy carrier.

Claude (C 146) and Guillain (G 178) have both reported possible infection by contact with chronic cases as described above under the heading: *"Cases occurring in hospitals or among doctors and nurses."*

Infection in the newborn.—Transmission of epidemic encephalitis from mothers to newborn babies has been described by Hall. The following is quoted from his book (H 29a):

"In the majority of cases in this country, and probably elsewhere, in which a pregnant woman suffering from encephalitis comes to full term, the child is healthy when born and remains so. On the other hand, a certain number of cases are on record in which the child born of an infected mother has itself suffered from the disease. Such infection may occur in utero, or after birth. There is also some evidence which suggests that infection may be conveyed by the mother's milk.

"As regards infection in utero, or 'placental transmission,' Levaditi, Harvier, and Nicolau [L 197] have shown that the virus obtained from fatal cases of encephalitis lethargica in man can, in rabbits, be transmitted through the placenta to the foetus in utero and localize itself in the foetal nervous system. Kononowa, in Moscow [quoted by Pappenheim (P 28)], found typical lesions of encephalitis lethargica chiefly about the third and fourth ventricles and around the aqueduct in the four-month foetus of a human case fatal during pregnancy. Novaes and Sousa [quoted by Jorge (J 60, J 61)] saw a case in which the baby of an infected mother had definite

myoclonus during delivery. In each of the above cases there must have been 'placental transmission.'

"In one of the earliest cases recorded in the United Kingdom, the patient was delivered of a full-term child, which was itself affected shortly after birth [Harris (H 74)]. In Mercier, Andrieux and Bonnaud's [M 263] case the mother was attacked twenty-two days before confinement; the child developed a mild attack in the second week. Santi [S 55] described a similar case in Italy, and Parsons [P 47a] refers to three further cases in this country. In such cases it is possible that the infection has not been by 'placental transmission,' but by direct contagion at or after birth. That such later infection may occur is shown by a case recorded by Hallè [H 40.] In his case the mother had become affected whilst suckling a four-month-old baby. In spite of her severe attack, she declined to allow the baby, then in excellent condition, to be weaned. Nine days after the onset of the mother's illness the child became affected, and died on the fifth day; the mother recovered. In this case there is the possibility that infection was conveyed by the mother's milk. This view has also been put forward by Klippel and Baruk, [K 82] on the following grounds: A pregnant woman began with epidemic encephalitis two weeks before term. She was delivered of a healthy full-term child which weighed 8½ pounds at birth and was lively and active. After being put to the breast, it gradually changed, becoming weaker, and eventually developed torpor and somnolence exactly like its mother's condition. After eight days it was weaned, and almost at once its vigour and activity returned. Since then its health has been excellent. The authors think that in this case the milk may have contained some attenuated poison which was the sole cause of the child's symptoms. Sterling [S 319] reports a case in Warsaw, in March, 1924, in which

there was evidence of direct contagion between the mother, the suckling, and the servant."

Bompiani (B 277) describes a case in Italy, and Shuman (S 173) one in Syria, in which infants were born of mothers suffering from epidemic encephalitis. Both of these babies died, one three days and the other thirty-six hours after birth, with symptoms suggestive of the same disease.

Addtional references to cases showing possible transmission of epidemic encephalitis from man to man, either directly or indirectly, are given below:

Alessandri (A 79)
Bériel, L. and Morenas (B 159)
Buss and Peltzer (B 446)
Buzzard, E. F. (B 454)
Buzzard and Greenfield (B 459
d'Arbela, F. and A. Montanari (D 31a)
Dopter, C. (D 155)
Draper, G. (D 165)
Ebaugh, F. G. (E 4)
Fernandez-Sanz, E. (F 28a)
Froment and Comte (F 143)
Kaneko and Aoki (K 14a)
Ladroitte, P. (L 14)
de Laroche (L 66)
Laubie (L 76)
Lépine (L 140)
Léri and Gay (L 154)
Lévy, P. (L 236)
Mäkelä, V. (M 29)
Manicatide (M 56)
Menninger, K. (M 260)
Mercio Xavier, A. (M 264)
Netter, A. (N 68)
Nixon, C. E. and T. H. Sweetser (N 114)
Pecori, G. (P 65)
Péju, G. and E. Abel (P 72)
Pfister, M. (P 130)
Pic, A. (P 136)
Pic, A. *et al.* (P 140)
Reich, H. (R 49)
Roger, H. and A. Blanchard (R 211)
Römer, C. (R 179)
Ronchetti, V. (R 230)
Salvetti, I. (S 39)
Sterling, W. (S 318)
Stern, F. (S 328)
Taddei, G. (T 2)

England and Wales

"The earliest characteristic case of lethargic encephalitis of which we have record (during the present epidemic) is stated to have had its onset in Bermondsey on February 11, 1918." (J 25.)

This statement was made by James in the *Local Government Board Reports*. However, thirteen cases, reported by Melland (M 253) as "epidemic polioencephalitis or so-called epidemic botulism," were probably lethargic encephalitis, although their true nature was not then understood. The onset of the first of this group was in November, 1917; two occurred in December; one in January, 1918; one in February, and the rest in March and April.

It has been suggested that the disease may have been introduced into England through cases which occurred on the British front in 1916, although they were not recognized as encephalitis at the time. Hall (H 29a) quotes the following record (Epidemic Encephalitis, 1924, p. 7.) "In June of 1916 a soldier was invalided home and came under my care at the Third Northern General Hospital, Sheffield, suffering from what was then put down as an 'atypical paralysis agitans.' He was only 38: the characteristic expression and posture, though slight in degree, were unmistakable." This case was followed up seven years afterward and it was concluded that the development was not typical of presenile paralysis agitans and that "the residua obviously resemble those of encephalitic origin."

However, the possibility of an independent focus in England must also be considered. James (J 25) says: "Dr. Gardner of Shrewsbury has stated his belief that in May, 1916, he attended a patient whose illness, in all possibility, was of the same nature."

1918.—Although this disease was not made reportable in England until January 1, 1919, we find in the *Local Government Board Report* n. s. 121, 1918, a complete "Report of an Inquiry into an Obscure Disease, Encephalitis Lethargica." Of 338 cases of illness examined, 126 were considered typical and were selected for study. They were distributed by months as follows:

Feb.	Mar.	Apr.	May	June	Total	Deaths
1	35	56	28	6	126	25

Table 1

Incidence and Mortality by Age

Age group	Percentage of cases	Percentage of deaths
Under 10 years	20.6	8
10–19	19.0	8
20–29	15.8	16
30–39	16.5	32
40–49	13.4	20
50–59	10.0	16
60–70	4.7	0
70 and over	0	?
	100.0	

From James: *Local Government Board Reports.*

Sex.—"There were more male patients than female patients, the respective numbers for the 126 cases . . . being 68 and 58."

Occupation and Social Status.—Among the adult patients there were nearly as many occupations represented as there

were cases of illness. There were two doctors, two nurses, and one undertaker. Social status covered a wide range.

1919 and 1920.—A portion of Table 1, page 260-A of the *Great Britain Ministry of Health, Annual Report of Chief*

Table 2

INCIDENCE AND MORTALITY BY AGE AND SEX [524 CASES]

Total number of known cases	Total number of deaths	Fatality per 100 cases	Under 1 year								1							
			Male				Female				Male				Female			
			Cases	Percentage of known cases	Deaths	Percentage of total deaths	Cases	Percentage of known cases	Deaths	Percentage of total deaths	Cases	Percentage of known cases	Deaths	Percentage of total deaths	Cases	Percentage of known cases	Deaths	Percentage of total deaths
524	286	54.6	4	0.8	4	1.4	4	0.8	2	0.7	11	2.1	7	2.4	20	3.8	16	5.6
			5								10							
			21	4.0	10	3.5	19	3.6	11	3.8	34	6.5	13	4.5	22	4.2	7	2.4
			15								20							
			33	6.3	16	5.6	40	7.6	20	7.0	34	6.5	15	5.2	42	8.0	24	8.4
			30 and over								All ages							
			113	21.6	68	23.8	127	24.2	73	25.5	250	47.7	133	46.5	274	52.3	53	53.5

From *Great Britain Ministry of Health, Annual Report of Chief Medical Officer.*

Medical Officer, 1919-20, has been copied below in Table 2. This table shows "the total number of cases and deaths [in 1919], the fatality per 100 cases, together with the age and sex distribution at certain age periods and the number of deaths in each group." This analysis was based on a total number of 524 known cases (corrected total 541).

Parsons (P 47a) has given a careful analysis of 1,273 cases occurring in England and Wales in 1919 and 1920. The following is taken from his report.

Sex.—There was no discrimination between the sexes. For this series, the figures showed 634 males and 639 females.

Table 3

Sex Incidence of Encephalitis Lethargica

Age group	Sex	
	Male	Female
0–1	6	6
1–5	30	44
5–10	48	47
10–15	82	69
15–20	89	87
20–30	83	108
30–40	91	102
40–50	103	84
50–60	65	56
60–70	33	24
70–80	4	2
80–90	0	1
Total	634	639

From Parsons (1,273 cases).

Table 4

Age Incidence of Encephalitis Lethargica

Age group	Cases
0–10	181
10–20	327
20–30	191
30–40	193
40–50	187
50–60	121
60–70	57
70–80	15
80–90	1
Total	1,273

From Parsons (1,273 cases).

Table 5

Quinquennial Periods 1-20 Years

0–1 Year			1–5 Years			5–10 Years			10–15 Years			15–20 Years		
Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage
12	9	75.0	74	50	67.5	95	45	47.3	151	50	33.1	176	74	42.0

DECENNIAL PERIODS 20-80 YEARS

20–30			30–40			40–50			50–60			60–70			70–80		
Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage	Cases	Deaths	Death Percentage
191	80	42.1	193	93	47.8	187	94	50.0	121	72	59.5	57	36	63.3	15	12	80.0

From Parsons: Report on encephalitis lethargica. *Great Britain Ministry of Health, Reports on Pub. Health and M. Subj.*, No. 11, 1922.

Table 6

INCIDENCE BY OCCUPATION

Occupations involving a considerable time being spent indoors I		Occupations involving a considerable time being spent out-of-doors II		Indeterminate group III	
Household duties	217	Labourers	54	Artisans and mechanics	96
School children	199	Farmers, gardeners, etc.	31	Professional	62
Factory hands	83	Travellers, postmen, messengers and others	24	No occupation	23
Shopkeepers	60	Seamen	11	Ex-service men	16
Clerks	60	Police constables	5	Various	6
Domestic servants	37	Medical men	4		
Tailoring	23	Vagabonds	3		
Miners	22	Dairymen	1		
School teachers	21				
Nurses	9				
Publicans	3				
Totals	734		133		203

From Parsons (1,070 cases).

Age incidence.—These statistics show that the disease is found chiefly among those approaching adult life; 25 per cent of the total series occurred among young people between the ages of ten and twenty years. (Table 4)

Mortality.—"The case mortality works out at 48.3 per cent." The figures for different age periods are given in Table 5 copied from Tables 48 and 49 of this report.

Occupation.—A classification by occupation was attempted in the case of 1,070 patients for whom information was available (Table 6).

The author concludes that the disease attacks chiefly those individuals who spend the greater part of the day indoors. Attention is also called to "the small number of medical men and nurses who have suffered from the disease, thus suggesting that contagion must play a very small aetiological part. The fact, too, that only one case was reported among dairy workers is not without significance when considering the question of possible avenues of infection."

During 1920, a small group of thirty-two cases was studied by McClure in Manchester. They were distributed throughout the year, although the greater number occurred in the winter months. They were widely spread over the city, and no connection was traced between any two of the cases. The ages ranged from infants to an adult of fifty-nine years. There was the same number of males and of females. Occupation and class seemed to have no bearing upon the incidence. The mortality rate was 53 percent.

1924.—The Sheffield Epidemic: The greatest incidence of encephalitis in England was in 1924. In Great Britain, the cities with the highest rates were Glasgow and Sheffield. In Glasgow there were 398 confirmed cases and in Sheffield 301. An intensive study of the Sheffield outbreak was conducted

by the Medical Research Council. The figures given in the Epidemiological Report by F. E. Wynne are quoted here.

This writer calls attention, as do many others, to the element of error in all figures dealing with encephalitis, as the "number of abortive and missed cases cannot be even approximately estimated."

Age and sex.—Tables 7 and 8 give "actual cases and rates of incidence in age periods for sex," and "actual deaths in age periods in both sexes." (Charts XXI and XXII.)

Table 7

CASES AND RATES OF INCIDENCE IN AGE PERIODS FOR SEX

Age periods, years	Males		Females		Total	
	Cases	Attack rate per 1,000	Cases	Attack rate per 1,000	Cases	Attack rate per 1,000
Under 5 years	3	0.116	11	0.436	14	0.275
5–9	25	0.966	23	0.878	48	0.922
10–14	26	0.965	15	0.548	41	0.754
15–19	34	1.402	23	0.890	57	1.138
20–24	19	0.922	15	0.630	34	0.765
28–34	30	0.795	14	0.336	44	0.554
35–44	17	0.454	11	0.287	28	0.369
45–54	14	0.459	14	0.478	28	0.468
55–64	14	0.808	3	0.169	17	0.485
65–74	3	0.375	2	0.210	5	0.286
Over 75 years	1	0.486	.....	.....	1	0.182
City	186	0.725	131	0.488	317	0.603

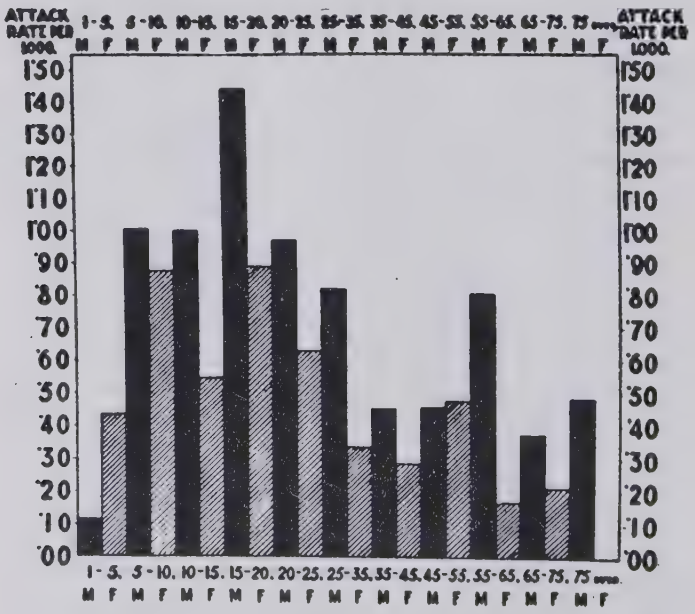
From Wynne: *The Sheffield Outbreak of Epidemic Encephalitis in 1924.*

Chart XXI

EPIDEMIC ENCEPHALITIS

Sheffield, 1924

Attack Rate Per 1,000 of Population at Certain Periods in Each Sex Age and Sex



Reproduced from "The Sheffield Outbreak of Epidemic Encephalitis in 1924," W 151.

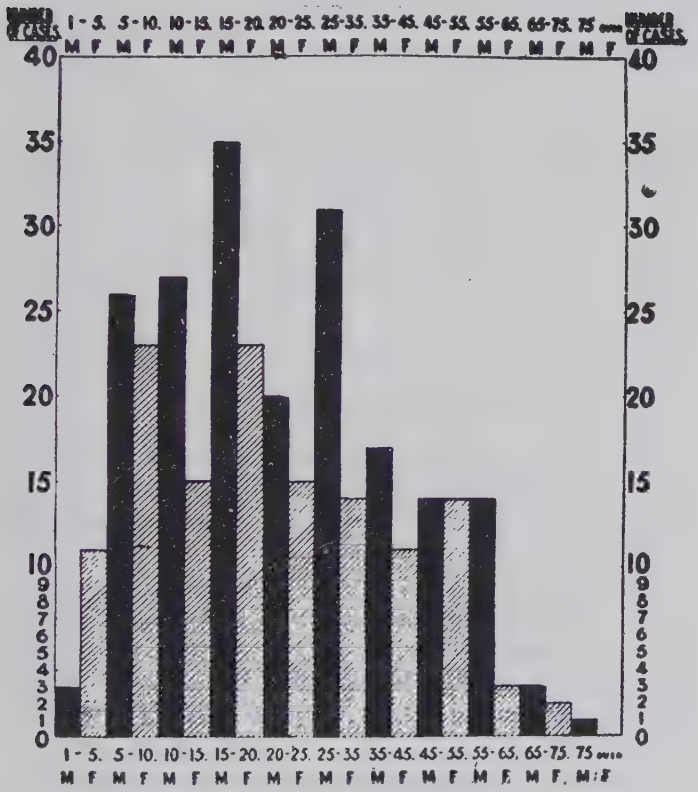
CHART XXII

EPIDEMIC ENCEPHALITIS

SHEFFIELD, 1924

AGE AND SEX DISTRIBUTION OF CASES

AGE AND SEX



Reproduced from "The Sheffield Outbreak of Epidemic Encephalitis in 1924," W 151.

Table 8

ACTUAL DEATHS IN AGE PERIODS IN BOTH SEXES

Age Period	Males	Females
Under 5	1	1
5–10	3	5
10–15	2	2
15–20	5	1
20–25	1	0
25–30	4	0
30–35	1	2
35–40	0	0
40–45	2	3
45–50	1	1
50–55	1	1
55–60	2	2
60–70	3	1
Total	26	19

From Wynne: *The Sheffield Outbreak of Epidemic Encephalitis in 1924*

The author notes an increased susceptibility to infection in males, and an increased liability to death in females, and also an increased susceptibility to infection in males between the ages of fifteen and thirty years.

Mode of Invasion.—"A study of the notifications received showed that from the commencement the outbreak was general throughout practically the whole city. Cases occurred sporadically in all parts simultaneously and there was no evidence of the march of any infection from district to district. . . . .

"The result of topographical study of this outbreak of en-

cephalitis, as of all others, are in fact negative, and everything points to a sudden and general lowering of resistance to a pre-existing and wide-spread virus."

Contagion.—In this outbreak, Professor Wynne was "able to find evidence of contact, more or less intimate, in six instances involving 13 patients, or just over 4 per cent. of the total number of cases." The particulars of these cases are given in the report.

A study of the occupations involved and of the social status of the patients led the writer to conclude that there was no relation between the disease and social conditions. The patients were classified by occupations as follows:

Domestic servants and married women	40
Metal workers	37
Miners	15
Unemployed	12
Laborers	10
Clerks	9
Warehouse men and storekeepers	8
Engineers	6
Turners	3
Plumbers	3
Errand boys	3
Printers	2
Confectioners	2
Window cleaners	2
Medical practitioners	2

The rest of the cases included one each among a large number of occupations.

The high incidence among metal workers and women en-

gaged in household duties was thought to be in due proportion to the adults so employed.

1925 and 1926.—"Since early in 1925, the number of cases [in England and Wales] has decreased slowly but steadily, apparently unaffected by seasonal influences." "It is noteworthy that the incidence of this disease has fluctuated extremely little in England in the course of 1926; the highest incidence was reported in February, when there were 212 cases, and the lowest (135) during the four weeks ending September 11th."

"Both case and death returns from England show that the disease is more prevalent in urban (6.3 cases per 100,000 in 1926) than in rural districts (4.8). Its incidence is highest in the north and decreases through the midlands to the south; it is lowest in Wales. This geographical distribution is the same for urban and rural districts." (The above statements are taken from the *League of Nations Monthly Epidemiological Reports.*)

The *Registrar General's Statistical Review* for 1926 gives an analysis of the deaths from epidemic encephalitis for that year by age and sex (Table 9).

Tables 10a and 10b give age, sex and seasonal incidence as noted in 524 cases reported in the English literature for the years 1917-26.

Compulsory notification was instituted by the Local Government Board beginning January 1, 1919. The available figures by years and months (or by four-week periods) are given in Tables 11 and 12.

Table 9

Mortality from Epidemic Encephalitis by Age and Sex [1926]

Ages at death	Male	Female
0–1	10	8
1–5	73	49
5–10	41	40
10–15	30	34
15–20	58	47
20–25	37	51
25–30	45	51
30–35	30	41
35–40	46	36
40–45	41	56
45–50	43	56
50–55	58	60
55–60	64	48
60–65	37	39
65–70	25	35
70–75	6	19
75–80	5	2
80 up	1	3
All ages	650	675

From the *Registrar General's Statistical Review*, 1926.

Table 10a

SEX AND AGE INCIDENCE OF EPIDEMIC ENCEPHALITIS

Year	Total	Sex		Age group										
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70 +	N.S.
1917	4	2	2	.....	.....	1	3							
1918	174	61	73	40	1	24	28	29	16	15	15	6	2	38
1919	79	30	49	.....	.....	11	27	14	6	6	5	2	...	8
1920	89	44	45	.....	.....	9	14	23	16	16	5	3	1	2
1921	24	15	9	.....	.....	0	6	4	5	5	2	...	...	2
1922	8	2	6	.....	.....	2	5	1						
1923	7	4	3	.....	.....	1	2	...	1	1	1	...	...	1
1924	43	27	13	3	.....	3	10	14	9	3	3	...	...	1
1925	3	2	1	.....	.....	...	1	...	1	1				
1926	4	2	2	.....	.....	...	...	1	1	1	...	1		
N.S.	89	33	28	28	5	24	12	21	11	5	3	3	1	4
Total	524	222	231	71	6	75	108	107	66	53	34	15	4	56

In 524 cases reported in the literature.

Table 10b

SEASONAL INCIDENCE OF EPIDEMIC ENCEPHALITIS

Year	I	II	III	IV	N.S.
1917	.....	.....	.....	3	1
1918	55	78	2	18	21
1919	19	12	13	25	10
1920	15	33	15	18	8
1921	18	4	0	1	1
1922	2	.....	1	5	
1923	4	.....	.....	1	2
1924	18	15	3	1	6
1925	.....	.....	1	.....	2
1926	2	1	.....	1	
N. S.	.....	.....	.....	.....	89
Total	133	143	35	73	140

In 524 cases reported in the literature.

Table 11

INCIDENCE AND MORTALITY OF EPIDEMIC ENCEPHALITIS BY YEARS [1918-27]

Year	Cases	Deaths			Case mortality, percentage
		M	F	Total	
1918	126	5	11	16	12.6
1919	541	138	156	294	54.3
1920	889	239	241	480	54.0
1921	1,470	351	378	729	49.6
1922	451	160	179	339	75.2
1923	1,023	276	255	531	51.9
1924	5,036	732	675	1,407	27.9
1925	2,634	666	706	1,372	52.1
1926	2,267	650	675	1,325	58.4
1927	1,617	.....	.....	1,155	71.4

From the *League of Nations Monthly Epidemiological Reports* and *Registrar General's Statistical Review* for 1926.

Table 12

Incidence of Epidemic Encephalitis by Year and Month [1918-28]*

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1918													
Cases	....	1	35	56	28	6	....	....	....	....	....	....	126
1919													
Cases		286			70			64			121		541
1920													
Cases		264			224			161			241		890
1921													
Cases	454	220	278	117	77	50	65	42	37	39	38	57	1,470
1922													
Cases	32	44	54	61	50	32	17	38	36	23	28	43	454
1923†													
Cases	66	151	184	145	96	73	54	29	36	52	50	53	49	1,038
1924													
Cases	56	150	397	806	1,066	862	477	266	236	187	173	172	219	5,067
1925													
Cases	194	231	261	206	294	257	207	173	159	166	186	165	154	2,635
1926													
Cases	208	212	179	201	185	176	183	159	135	151	152	180	152	2,267
1927													
Cases	138	159	157	129	194	131	117	110	100	142	106	134	1,617
1928‡													
Cases	....	....	....	....	....	....	....	....	....	....	....	....	742

From *Local Gov. Bd. Rep. N. S.* 121 *Sect. of Pub. H. Rep.* 36; *League of Nations Monthly Epidemiological Reports.*

* It has been thought best to give for the yearly totals the figures as they are most frequently reported, although it will be noted that these do not in every case correspond to the totals computed by adding together the figures given for the months.

† 1923-26 reported in four-week periods.

‡ To June 30.

Wales.—"A case was reported from Bangor early in 1918, but it is in South Wales that the disease has chiefly declared itself, and particularly in the country of Glamorganshire. The incidence corresponds closely with the density of population and it may be added that a large proportion of the Glamorganshire mountainous regions of Wales have remained exempt." Notified cases, 1919, 25; 1920, 46. (Report on Public Health and Medical Subjects, No. 11, 1922).

Scotland

The first recorded case of epidemic encephalitis in Scotland was observed by Findlay in Glasgow, in March, 1918 (F 42). Picken (P 145) reported a case with onset in April, 1918. Brownlie (B 408) described another in Glasgow in May, and Findlay saw two cases in June of the same year. Chalmers (C 83), Picken (P 145) and McLean (M 230) mention eleven cases with five deaths occurring in and around Glasgow in the spring of 1918; "subsequent information added to the number."

Parsons (P 47a) commented that encephalitis lethargica was notifiable in Aberdeen Burgh only, and thus it was difficult to say to what extent it prevailed in Scotland. "From published reports and such other information as is available, it seems as if the disease, as elsewhere in Britain, is chiefly reported from the larger centers of population, and more especially from Edinburgh and Glasgow."

Bramwell and Miller (B 366) studied fifty-seven cases, seventeen of which were in Edinburgh (Table 1).

Table 1

INCIDENCE BY MONTH OF 57 CASES

1918		1919–20	
Mar.	5	Jan.	10
Apr.	4	Feb.	5
May	1	Mar.	2
June	3	Oct.	1
Aug.	1	Nov.	2
Oct.	2	Dec.	5
Nov.	2	Jan.	3
Dec.	8	Feb.	3
	26		31

From Bramwell and Miller.

Sex: 29 males and 28 females.

Ages: 17 to 75 years; deaths, 9; ages at death, 17, 18, 24, 27, 32, 42, 45, 47 and 60.

Findlay and Shiskin (F 43) reported 24 cases in children, November, 1919, to August, 1920: ages, 4 weeks to 15 years; males, 19; females, 5; deaths, 3.

Grace Anderson (A 104) describes forty cases of encephalitis lethargica in children with seven deaths.

Chalmers and Macgregor (C 86) note that encephalitis lethargica was made reportable in Glasgow in 1918; they give the admissions to two hospitals in Glasgow by quarters, 1918-22 (Table 2).

These authors reported 38 cases with 8 deaths: December, 1922, 1 case; January, 1923, 5 cases; February, 1923, 32 cases.

Ages 1½ to 56 years. Twenty-two of the 38 cases were over 15 years.

Table 2

ADMISSIONS TO TWO HOSPITALS, GLASGOW [1918-22]

Quarter	1918	1919	1920	1921	1922
I	.....	6	6	2	1
II	4	3	22	5	4
III	3	1	3	1	
IV	.....	11	3	4	

From Chalmers (C 84).

The epidemic as seen in Glasgow 1919-24 has been reviewed by Chalmers (C 84).

The recorded cases subsequent to 1918 (Glasgow) are given in Table 3. The mortality rate was 24 percent.

Table 3

RECORDED CASES OF ENCEPHALITIS IN GLASGOW [1919-24]

Year	Cases	Deaths
1919	.....	13
1920	61	20
1921	28	11
1922	20	10
1923	110	26
1924	398	46
Total	617	126

From Chalmers (C 84).

1923.—Of the reported cases, 73 were accepted as encephalitis lethargica (Table 4). There were 16 deaths. Of a total of 75 cases (including 2 from November, 1922) there were 39 males and 36 females.

Table 4

INCIDENCE BY MONTHS OF 73 CASES IN 1923

Month	Cases
Jan.	9
Feb.	34
Mar.	15
Apr.	4
May	3
June	2
July	2
Aug.	3
Sept.	1
Total	73

From Chalmers (C 84).

1924.—Of 530 notified cases, 398 were verified (Table 5).

Table 5

Incidence and Mortality of 398 Cases in 1924

Quarter	Cases		Deaths	
	Male	Female	Male	Female
I	1	3	1	
II	194	114	13	17
III	42	20	4	5
IV	12	12	4	2
Total	249	149	22	24

From Chalmers (C 84).

Incidence by months:

January	0	July	19
February	1	August	9
March	3	September	6
April	54	October	3
May	204	November	7
June	85	December	—

The age distribution of all notifications in Glasgow for 1919-24 is given in Table 6.

Table 6

AGE DISTRIBUTION, GLASGOW, ALL NOTIFICATIONS
[1919-24]

Age	Cases		Deaths		Morbidity percentage		
	Male	Female	Male	Female	Male	Female	Both sexes
— 5	23	11	6	9	26	82	44
—10	50	28	4	2	8	7	8
—15	74	27	4	6	5	22	10
—25	97	87	11	11	11	13	12
—35	36	37	8	13	22	35	29
—45	50	22	11	10	22	45	29
—55	27	21	12	9	45	43	44
—65	15	7	4	4	27	57	36
—75	2	1	1	1	50	100	67
75+	1	1	.....	.....	.....	.....	.....
All ages	375	242	61	65	16	27	20

The age, sex and seasonal distribution of 626 cases in Scotland as collected from the literature are shown in Tables 7a and 7b.

Table 7a

AGE AND SEX INCIDENCE OF 626 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age groups						
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	N.S.
1918	32	4	2	26	.....	2	2	.....	2	.....	26
1919	30	4	1	25	.....	1	.....	2	1	1	25
1920	38	23	9	6	3	14	10	1	2	2	6
1921	2	2	.....	.....	.....	1	.....	.....	1		
1922	3	.....	.....	3	.....	.....	.....	.....	.....	.....	3
1923	118	41	40	37	.....	3	3	1	1	.....	110
1924	402	252	150	.....	.....	.....	2	.....	.....	2	398
N.S.	1	1	.....	.....	.....	1					
Total	626	356	230	97	3	22	17	4	7	5	568

Table 7b

SEASONAL INCIDENCE OF 626 CASES REPORTED IN THE LITERATURE

Year	Quarter				
	I	II	III	IV	N.S.
1918	6	13	1	12	
1919	17	2	.....	11	
1920	7	24	7		
1921	2	.....	.....		
1922	.....	.....	.....	3	
1923	102	10	6		
1924	4	308	66	24	
N.S.	.....	.....	.....	.....	1
Total	138	357	80	50	1

The reported cases and deaths in Scotland (16 cities) for 1921-27 are given in the *League of Nations Monthly Epidemiological Reports* (Table 8).

Table 8

Year	1921	1922	1923	1924	1925	1926	1927
Cases	.....	.....	.....	631	206	194	130
Deaths	61	45	125	181	154	170	117

The death rate for the period 1921-26 (per 100,000) was 2.5 percent.

The figures in Table 9, giving the distribution by months (or four-week periods) for 1923-28, have been taken from the *League of Nations Monthly Epidemiological Reports.*

Table 9

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN SCOTLAND
[1923-28]

Month	1923	1924*	1925	1926	1927	1928
Jan.	.....	3	22	22	11	10
Feb.	.....	2	28	19	9	9
Mar.	.....	5	28	26	17	9
Apr.	13	8	22	12	15	10
May	11	95	20	12	15	16
June	4	232	22	15	5	11
July	6	144	2	10	11	
Aug.	10	46	12	5	11	
Sept.	3	33	14	12	7	
Oct.	2	20	16	11	19	
Nov.	2	10	21	17	2	
Dec.	3	22	4	15	5	
		16				
Total	54	636	211	168	127	

* By four-week periods.

From the *League of Nations Monthly Epidemiological Reports.*

IRELAND

The available records of epidemic encephalitis in Ireland are few in number and are confined chiefly to Dublin and Belfast.

In Dublin, a case was observed in April, 1918, by O'Carroll

and Nesbitt (O 7), another in August of the same year, and two in January, 1919.

Sir John Lumsden (L 351) described one case of encephalitis lethargica with onset in November, 1919, and stated that twenty or thirty cases had been observed in Dublin, but gave no details with regard to the others.

Parsons (P 47a) gives the incidence in the north of Ireland: 1918, 1; 1919, 2; 1920, 28; 1921, 6. He remarked "that nearly all the patients from the districts outside of Belfast were connected by business with the city or had visited it shortly before their illness. It is in, and around, the chief industrial center of Ireland, then, that most of the reported cases of encephalitis have occurred."

The Belfast epidemic was studied by Marshall (M 141). In the County Borough of Belfast deaths were reported as follows:

1918	1919	1920	1921	1922	1923	Total
3	7	24	5	3	20	62

Up to June 21, 1924, there had been 211 cases in Belfast with 17 deaths.

The author made a study of the records of 42 patients at the Royal Victoria Hospital from October 5, 1918, to December 31, 1923: mortality, 33⅓ percent; males 30, with 11 deaths; females 12, with 3 deaths. Two others died after leaving the hospital, but the cause of death was not determined. The incidence by age, sex and season is given in Table 1.

Table 1

AGE, SEX AND SEASONAL INCIDENCE OF EPIDEMIC ENCEPHALITIS [BELFAST]*

Year	Cases	Sex		Age group						Quarter			
		Male	Female	0–10	10–20	20–30	30–40	40–50	50–60	I	II	III	IV
1918	1	.....	1	...	...	...	...	1	...	...	...	...	1
1919	2	1	1	...	1	1	...	...	...	1	...	...	1
1920	19	15	4	...	4	7	3	3	2	1	17	1	
1921	3	2	1	...	2	...	1	...	...	1	2		
1922	1	.....	1	...	...	...	...	...	1	...	...	1	
1923	16	12	4	1	4	1	4	3	3	8	4	4	
Total	42	30	12	1	11	9	8	7	6	11	23	6	2

* 42 patients, Royal Victoria Hospital.

Thus the most marked incidence was in April, May and June. No evidence of contagion could be determined in any of these cases.

Northern Ireland: Cases and deaths by years are reported in the *League of Nations Monthly Epidemiological Reports* and *Report on Public Health,* No. 11, 1922 (Table 2).

Table 2

EPIDEMIC ENCEPHALITIS, INCIDENCE AND MORTALITY
[1918-27]

Year	Cases	Deaths
1918	1	
1919	2	
1920	28	26
1921	6	19
1922		5
1923	23	34
1924	278	80
1925	23	26
1926	21	38
1927	12	28

From the *League of Nations Monthly Epidemiological Reports* and *Reports on Public Health,* No. 11, 1922.

The distribution by four-week periods or by months is given for 1923-26 in Table 3.

Table 3

INCIDENCE OF EPIDEMIC ENCEPHALITIS [1923-26]

Month	Northern Ireland		Irish Free State	
	1923*	1924*	1925	1926
Jan.	0	1	1	1
Feb.	2	1	1	0
Mar.	2	0	1	0
Apr.	5	1	4	0
May	2	124	3	1
June	2	71	1	0
July	3	13	2	0
Aug.	0	4	0	1
Sept.	2	5	1	0
Oct.	1	0	1	0
Nov.	1	1	0	0
Dec.	0	1	1	0
	0	0		
Total	20	222	16	3

* Figures given by four-week periods.

From the *League of Nations Monthly Epidemiological Reports* and *Reports on Public Health*, No. 11.

An analysis of fifty-six cases reported in the literature is given in Tables 4a and 4b.

Table 4a

AGE AND SEX INCIDENCE OF 56 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group						
		Male	Female	1–9	10–19	20–29	30–39	40–49	50–59	60–69
1918	4	1	3	.....	.....	2	.....	1	1	
1919	6	3	3	.....	2	2	.....	1	.....	1
1920	19	15	4	.....	4	7	3	3	2	
1921	5	2	3	1	2	.....	1	.....	1	
1922	1	.....	1	.....	.....	.....	.....	.....	1	
1923	16	12	4	1	4	1	4	3	3	
1924	4	3	1	.....	2	.....	.....	1	1	
N.S.	1	1	.....	.....	.....	.....	.....	.....	.....	1
Total	56	37	19	2	14	12	8	9	9	2

Table 4b

SEASONAL INCIDENCE OF 56 CASES REPORTED IN THE LITERATURE

Year	I	II	III	IV	N.S.
1918	.....	1	1	2	
1919	3	.....	.....	3	
1920	1	17	1		
1921	2	3	.....		
1922	.....	.....	1		
1923	8	4	4		
1924	2	2	.....		
N.S.	.....	.....	.....	.....	1
Total	16	27	7	5	1

France

In April, 1917, under the title "Quarante cas d'encéphalomyélite subaiguë," Cruchet, Moutier and Calmettes published an account of the first cases of encephalitis observed as a group during this epidemic (C 314). These cases are frequently referred to as having occurred at Verdun in the winter of 1915-16, although Cruchet was not specific as to location or dates in his first communication. Later articles (C 315, C 313, C 309) report these same cases, with some additions in more detail, making sixty-four cases in all. The diagnosis of two cases at Commercy, one in February, and one in September, 1915, is considered somewhat doubtful by the author; five cases at Verdun (June and December, 1915, January and February, 1916) are included in the group because the later appearance of cases of encephalitis led the author to believe that these may have been the same. At Bar-le-Duc, forty-eight cases developed in the interval from March, 1916, to May, 1917, distributed as follows:

1916	March	May	July	Aug.	Sept.	Oct.	Nov.	Dec.
	1	1	1	1	4	4	4	7

1917	Jan.	Feb.	Mar.	Apr.	May
	5	7	7	3	3

The mortality rate was 37.5 percent.

These cases were confined to the same regiment or to neighboring regiments, and did not spread. Cruchet saw none in the civil population at this time.

Twelve cases, diagnosed as poliomyelitis, were described

by Etienne in 1917: "Une épidémie militaire de myelitis aiguës" (E 57, E 58). Two cases occurred in August, 1915, and ten in May, 1916.

There were no other cases either before or after them, either in the army or among the civilian population. Etienne's description of the course of the illness raises the question whether these were cases of poliomyelitis or of epidemic encephalitis.

Gutmann and Kudelski (G 210) observed progressive symptoms in a former soldier at Champagne and made a retrospective diagnosis of epidemic encephalitis, with the acute illness in March, 1916.

Another chronic case reported by Vignard, Mouriquand and Léorat (V 75) from Lyon, was thought to have had its onset in August, 1917.

The first case of epidemic encephalitis in Bordeaux and the region of the southwest occurred at Libourne in March, 1918; there were three at Jouzac, March to April, 1918; three at Bordeaux, February to August, 1919; and twenty in the winter of 1919-20 in or near Bordeaux as reported by Cruchet and others (C 295, C 313, and C 320). In all, Cruchet saw 145 cases in the Bordeaux region between March, 1918, and October, 1920. He felt that the disease was probably carried to Bordeaux by soldiers, although cases of evident contagion could not be pointed out, even among those sleeping together. Twenty-seven died, a mortality of 18.6 percent.

In March, 1918 Netter also observed seven patients with encephalitis lethargica in Paris (N 44), and reported them together with a case of Dr. Lereboullet; four of this number died. At about the same time, other cases were reported in Paris by Claude (C 130); Sainton (S 21); de Saint-Martin and Lhermitte (S 19); and Chauffard and Bernard (C 107).

During the first week of January, 1920, Netter (N 47), called the attention of the Academy to a recrudescence of lethargic encephalitis and cited twelve cases that he had seen between November 26 and December 30, 1919.

Soon afterward Combemale and Duhot (C 181a) reported five cases from Lille. The first patient, a soldier, became lethargic three days after his return from Algiers in September, 1919. Two other cases developed in December, 1919, and two in January, 1920.

During the winter of 1919-20, a group of twenty-seven cases was observed in the Voges by Péju and Abel (P 72). The mortality was 55 percent. These authors believed the incubation period to be rapid, from thirty to forty-eight hours. They observed possible direct contagion on two occasions; one between husband and wife, and the other between two soldiers who slept next to each other.

Early in 1920, the disease assumed epidemic proportions. Bernard and Renault (B 168) gave an account of an inquiry instituted by the French Ministry of Health. Four hundred and sixty-four cases were reported from January 1 to May 1, 1920:

Jan.	Feb.	Mar.	Apr.	May	June
70	206	144	44	5	1

One hundred and eighteen deaths were certified, a mortality of 30 percent. Adults were affected twice as frequently as children, but the average mortality was the same in both periods of life. There were five cases in persons over sixty years of age.

In March, 1920, Netter (N 50) estimated that, up to that

time, there had been 10,000 cases if encephalitis lethargica in all of France, 1,500 of these in Paris. He himself saw 177 cases from November 1, 1919, to October 31, 1920 (N 55, N 56, N 71). More than half (92) occurred during the winter months, December, January and February. The age distribution is given in 50 cases and the sex in 70 cases.

Table 1

AGE, SEX AND SEASONAL DISTRIBUTION OF 177 CASES [NETTER]

1919	Cases	Male	Female	Age group	Cases
Nov.	7	29	41	Less than 10 years	6 (one 2 mo.)
Dec.	32	.....	.....	10–20 "	6
1920					
Jan.	26	.....	.....	20–30 "	9
Feb.	34	.....	.....	30–40 "	16
Mar.	25	.....	.....	40–50 "	7
Apr.	24	.....	.....	50–60 "	4
May	12	.....	.....	Over 60 "	2 (one 68
June	7	.....	.....	.....	and one 87)
July	5				
Aug.	1				
Sept.	1				
Oct.	3				
Total	177	29	41	.....	50

Out of 174 cases, which Netter had observed, there were, he believes, eight clear cases of contagion (4.6 percent).

One thousand, seven hundred and sixty-seven cases reported in the French literature have been analysed in Tables 2a and 2b.

Table 2a

Age and Sex Incidence of 1,767 Cases Reported in the Literature

Year	Total	Sex			Age group									
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S
1915	5	5	.....	.....	.....	...	...	2	1	1	...	...	....	1
1916	25	25	.....	.....	.....	...	...	5	8	12				
1917	28	28	.....	.....	.....	...	...	7	10	10	...	...	....	1
1918	98	49	49	.....	.....	2	18	24	20	12	9	6	....	7
1919	251	143	106	2	.....	9	54	75	43	23	20	6	....	21
1920	840	456	375	9	5	71	221	234	109	81	44	16	3	56
1921	106	52	53	1	1	10	17	32	15	16	3	4	....	8
1922	63	39	24	.....	.....	6	9	22	15	9	1	...	....	1
1923	69	39	30	.....	.....	7	17	20	11	7	5	...	....	2
1924	43	24	18	1	.....	3	7	12	7	6	4	3	....	1
1925	33	16	17	.....	.....	2	7	10	1	3	5	1	1	3
1926	13	4	9	.....	.....	3	2	3	3	...	2			
1927	3	1	2	.....	.....	...	1	2						
N.S.	190	102	84	4	3	21	39	41	24	20	12	4	6	20
Total	1,767	983	767	17	9	134	392	489	267	200	105	40	10	121

Table 2b

SEASONAL INCIDENCE OF 1,767 CASES REPORTED IN THE LITERATURE

Year	Quarter				
	I	II	III	IV	N.S.
1915	1	1	1	2	
1916	3	1	6	15	
1917	19	6	3		
1918	41	20	9	15	13
1919	68	16	11	132	24
1920	542	130	29	85	54
1921	66	6	9	5	20
1922	9	10	5	29	10
1923	48	8	1	5	7
1924	23	5	5	5	5
1925	12	6	3	8	4
1926	7	3	1	2	
1927					3
N.S.					190
Total	839	212	83	303	330

SWITZERLAND

The onset of the first recognized case of encephalitis lethargica in Switzerland was in December, 1917. The patient was evidently seen by both Bing (B 225) and Staehelin (S 300) in Basle, and is described by each of them.

During the pandemic of influenza in Switzerland in 1918-19, "l'encéphalite est restée silencieuse" (Carrière), only to break out in the first few months of 1920 with the largest number of cases for any year of this epidemic period.

During 1920, 984 cases were registered in Switzerland with a morbidity of 2.5 cases for 10,000 inhabitants. Dr. Carrière (C 48) gives an analysis of this epidemic. Compulsory registration did not go into effect until February 17; hence the reported total is probably incomplete. It is noted that 826 cases were registered during January, February and March of this year. The figures in Tables 1, 2 and 3 are taken from Dr. Carrière's report.

Table 1

Epidemic Encephalitis in Switzerland [Carrière] 1920

Month	Cases	Deaths
Jan.	38	16
Feb.	440	111
Mar.	348	96
Apr.	78	29
May	25	11
June	11	12
July	12	4
Aug.	6	2
Sept.	8	4
Oct.	4	3
Nov.	7	1
Dec.	7	1
Total	984	290

The mortality rate was 29.4 percent.

Of 984 notifications, the age and sex could be determined exactly in 528.

Table 2

AGE AND SEX DISTRIBUTION OF 528 CASES [CARRIÈRE]

Age group	Male		Female	
	Cases	Percentage	Cases	Percentage
Under 1 yr				
1–4 yrs.	5	1.8	7	2.8
5–14 "	46	16.5	35	14.0
15–19 "	46	16.5	34	13.6
20–29 "	45	16.2	59	23.6
30–39 "	48	17.3	45	18.0
40–49 "	44	15.8	36	14.4
50–59 "	22	7.9	17	6.8
60–69 "	17	6.2	13	5.2
70+"	5	1.8	4	1.6
Total	278	100.0	250	100.0

This author states that contagiousness must be slight, but remarks that "some familial or home epidemics were observed which leave no doubt of the contagious nature of the disease." No specific instances are given, however.

Naville studied the epidemic of encephalitis in Geneva, from 1918 to 1921, with especial reference to the sequellae. According to this author, 100 cases with 20 deaths had been pointed out in Geneva by Dr. Wartmen, a public health officer. But Naville saw 23 cases with sequellae that had not been diagnosed as encephalitis during the acute stage. Taking into consideration possible errors in diagnosis and neglect in the reporting of cases, this author concludes that there must have been from 150 to 200 cases in Geneva during the period from 1918 to 1921.

Table 3

AGE AND SEX DISTRIBUTION OF 290 DEATHS [CARRIÈRE]

Age group	Male	Female	Total
Under 1 yr.	1		1
1–4 yrs.	4	5	9
5–14 "	14	13	27
15–19 "	18	11	29
20–29 "	24	21	45
30–39 "	26	15	41
40–49 "	28	24	52
50–59 "	20	15	35
60–69 "	18	19	37
70+ "	9	5	14
Total	162	128	290

The Tables 4a and 4b give an analysis of 1,087 cases reported in the literature.

Table 4a

AGE AND SEX INCIDENCE OF 1,087 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age group									
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70	N.S.
1917	1	1	.....	.....	.....	...	...	...	...	1				
1918	11	5	6	.....	1	2	2	3	1	2				
1919	54	31	23	.....	.....	3	17	7	9	9	6	3		
1920	984	278	250	456	.....	173		104	93	80	39	30	9	456
1921	13	6	7	.....	1	3	1	2	4	2				
1922	4	1	3	.....	.....	...	3	1						
1923	7	4	3	.....	.....	...	3	1	1	2				
1924	3	3	.....	.....	.....	...	2	...	...	...	1			
1925	2	2	.....	.....	.....	...	...	...	...	1	1			
1926	2	2	.....	.....	.....	...	1	...	...	1				
1927	1	.....	1	.....	.....	...	...	1						
N.S.	5	3	2	.....	.....	...	2	2	...	...	1			
Total	1,087	336	295	456	2	212		121	108	98	48	33	9	456

Table 4b

SEASONAL INCIDENCE OF 1,087 CASES REPORTED IN THE LITERATURE

Year	I	II	III	IV	N.S.
1917	.....	.....	.....	1	
1918	2	.....	1	4	4
1919	11	4	2	12	25
1920	826	114	26	18	
1921	6	.....	1	2	4
1922	1	1	1	1	
1923	7				
1924	1	.....	.....	1	1
1925	.....	.....	1	1	
1926	.....	1	1		
1927	1				
N.S.	.....	.....	.....	.....	5
Total	855	120	33	40	39

From the *Bureau fédéral de Statistique* and the *League of Nations Monthly Epidemiological Reports,* we have Tables 5 and 6 showing incidence by years and months, 1920-27.

Table 5

INCIDENCE AND MORALITY OF EPIDEMIC ENCEPHALITIS BY YEARS [1920-27]

Year	Cases	Deaths	Death rate per 100,000
1920	984	290	7.5
1921	156	79	2.0
1922	62	42	1.1
1923	203	103	2.5
1924	87	78	2.0
1925	71	70	1.8
1926	36	55	
1927	23	44	
1928*	21		

* To July only.

From the *Bureau fédéral de Statistique* and the *League of Nations Monthly Ebidemiological Reports.*

Table 6

INCIDENCE OF EPIDEMIC ENCEPHALITIS BY MONTHS
[1920-27]

Month	1920	1921	1922	1923	1924	1925	1926	1927
Jan.	38	28	.....	52	2	4	2	4
Feb.	440	49	.....	68	11	11	1	3
Mar.	348	36	.....	41	12	21	5	3
Apr.	78	.....	.....	9	14	11	2	3
May	25	.....	.....	8	17	10	3	3
June	11	.....	.....	7	8	3	3	0
July	12	.....	.....	2	5	5	4	0
Aug.	6	.....	.....	3	7	1	1	0
Sept.	8	.....	.....	0	4	2	6	2
Oct.	4	.....	.....	4	0	0	2	1
Nov.	7	.....	6	5	1	0	2	0
Dec.	7	.....	5	4	4	3	5	3
Total	984	156	62	203	85	71	36	22

From the *Bureau fédéral de Statistique* and the *League of Nations Monthly Epidemiological Reports.*

Italy

D'Arbela and Montanari (D 31a) refer to cases of encephalitis lethargica in Salerno in the winter of 1918 as the first observed in Italy, followed by thirty typical cases in Prato (Micheli) in the spring. In Florence, eight cases were reported in 1918, one each in January, February and May, and five in the second half of the year.

Late in 1919 more cases began to appear in widely separated districts, followed by a tremendous outbreak in 1920, with a total of 5,009 cases reported in all Italy for the year.

In Turin, Almasio reported 138 cases, with 48 deaths (34 percent) distributed as shown in Table 1.

Table 1

Age, Sex and Seasonal Incidence of 138 Cases [Almasio, Turin]

Age group	Cases	Deaths	Male	Female	Month	Cases
Under 5 yrs.	4	2			1919	
5–10 yrs.	5	1			Dec.	4
10–20 "	19	4			1920	
20–30 "	47	18			Jan.	74
30–40 "	31	7			Feb.	43
40–50 "	15	9			Mar.	17
50–60 "	6	1				
60–70 "	7	3				
70–80 "	4	3				
Total	138	48	74	64		138

Pecori (P 65) describes the epidemic in Rome. This started with 80 cases in December, 1919, and 108 cases in January, 1920. Of 338 cases among civilians, there were 158 male and 180 female, with 91 deaths. Of nine soldiers affected, two died. The age distribution is shown in Table 2.

Table 2

AGE DISTRIBUTION OF 338 CASES AND 91 DEATHS
[PECORI, ROME]

Age group	Cases	Deaths	Mortality percentage
Under 1 yr.....	1	0	0
1–5 yrs.....	14	3	21.4
6–10 "	18	2	11.1
11–20 "	89	20	22.3
21–40 "	124	35	28.2
41–60 "	78	27	34.6
Over 60 "	14	4	28.5
Total.....	338	91	

The professions varied; 11 percent of the women were servants. Eight cases of possible contagion are cited. Judging from these, the author thought the incubation period was four to fifteen days.

Pecori also refers to 117 cases which occurred in Milan or nearby communities during the period January-May, 1920. Ronchetti (R 230) describes more fully 20 of these cases (all women), with 7 deaths; in the age period 15-20 years, 4 cases; 21-25 years, 4; 25-30 years, 2; 30-35 years, 3; 35-40 years, 3; 40-45 years, 3; and 60 years, 1 case. There were 10

housemaids, 4 charwomen, 4 factory workers, 1 dressmaker, and 1 manager of an asylum. Several cases occurred in one family.

On the island of Sardinia, 152 cases were examined by Rossi during the years 1919-23, (R 263). A map is given showing the location of the cases. This author never saw two in the same family.

D'Arbela and Montanari (D 31a) have reported on the incidence of encephalitis in Florence 1918-26 (Table 3).

Table 3

AGE AND SEX DISTRIBUTION OF 132 CASES [D'ARBELO AND MONTANARI, FLORENCE]

Year	Male	Female	Total	Age group	Percentage of cases
1918.....	5	3	8	Up to 10 yrs.	1.7
1919.....	7	3	10	10–20 "	25.8
1920.....	28	23	51	20–30 "	24.1
1921.....	7	3	10	30–40 "	18.9
1922.....	17	6	23	40–50 "	18.9
1923.....	5	7	12	50–60 "	8.6
1924.....	7	4	11	60–70 "	1.7
1925.....	4	2	6	70–80 "	0
1926.....	1	0	1		
Total....	81	51	132		

The mortality rate was 27 percent.

Of 60 cases observed by these authors, there were 38 males, with 36 deaths (22.4 percent).

One case of probable contagion from mother to child was noted. The greatest number of cases were from the working and peasant classes.

The age, sex and seasonal incidence of 877 additional cases collected from the Italian literature are given in the Tables 4a and 4b.

Table 4a

AGE AND SEX INCIDENCE OF 877 CASES REPORTED IN THE LITERATURE [ITALY]

Year	Total	Sex			Age group									
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.
1915	1	.....	1	.....	.....	...	...	1						
1916	1	1	.....	.....	.....	...	1							
1917	1	.....	1	.....	.....	...	...	...	1					
1918	12	9	3	.....	.....	1	...	4	3	2	...	2		
1919	139	73	63	3	3	12	33	40	19	9	5	...	...	18
1920	470	254	198	18	1	27	121	110	95	55	31	6	2	22
1921	56	29	24	3	.....	4	16	13	12	7	2	...	...	2
1922	52	28	20	4	.....	1	13	18	10	8	...	...	...	2
1923	10	2	6	2	.....	2	3	...	5					
1924	21	11	7	3	2	8	2	4	1	1	...	2	...	1
1925	10	3	6	1	.....	1	1	5	2	...	1			
1926	2	2	.....	.....	.....	...	...	1	1					
1927	1	.....	1	.....	.....	...	1							
N.S.	101	47	47	7	.....	7	24	29	21	9	7	2	...	2
Total	877	459	377	41	6	63	215	225	170	91	46	12	2	47

Table 4b

SEASONAL INCIDENCE OF 877 CASES REPORTED IN THE LITERATURE [ITALY]

Year	Quarter				
	I	II	III	IV	N.S.
1915	.....	.....	1		
1916	.....	.....	1		
1917	.....	.....	.....	.....	1
1918	.....	.....	.....	4	8
1919	20	5	8	78	28
1920	355	19	12	29	55
1921	27	5	3	8	13
1922	30	7	2	2	11
1923	4	3	.....	2	1
1924	8	4	1	1	7
1925	4	2	.....	.....	4
1926	2				
1927	1				
N.S.	.....	.....	.....	.....	101
Total	451	45	28	124	229

Nineteen cases from Sicily were reported in the literature for 1919-22 as shown in Table 5.

Table 5

AGE, SEX AND SEASONAL INCIDENCE OF 19 CASES REPORTED IN THE LITERATURE [SICILY]

Year	Total	Sex		Age group				Quarter				
		Male	Female	10–19	20–29	30–39	40–49	I	II	III	IV	N.S
1919	2	.....	2	.....	.....	2	.....	2				
1920	14	12	2	1	5	4	4	11	2	....	....	1
1921	2	1	1	1	1	.....	.....	2				
1922	1	1	.....	.....	.....	.....	1	1				
Total	19	14	5	2	6	6	5	16	2	....	....	1

The incidence in Italy by years, 1920-27, as given by the *League of Nations Monthly Epidemiological Reports,* is shown in Table 6.

Table 6

INCIDENCE OF EPIDEMIC ENCEPHALITIS BY YEARS [1920-27, ITALY]

Year	1920	1921	1922	1923	1924	1925	1926	1927
Cases.....	5,009	278	477	277	619	681	450	277

Other numbers of the *League of Nations Monthly Epidemiological Reports* give the incidence in four-week periods for 1923-25 and by months for 1926-28 (Table 7).

Table 7

Incidence of Epidemic Encephalitis by Four-Week Periods or Months [1923-28, Italy]

4-week periods				Months			
				1926		1927	1928*
1923 Cases	1924 Cases	1925 Cases		Cases	Deaths*	Cases	Cases
15	28	38	Jan.....	45	56	27	23
35	52	72	Feb....	39	47	29	26
70	150	92	Mar....	60	75	36	21
45	151	72	Apr....	66	50	28	15
27	72	66	May....	51	44	32	
15	37	42	June....	47	47	20	
8	18	31	July....	31	52	24	
6	21	29	Aug....	28	41	27	
4	13	15	Sept....	19	33	8	
7	15	14	Oct....	20	35	18	
15	12	13	Nov....	22	39	13	
15	22	20	Dec....	22	45	15	
15	27	19					
277	618	523		450	564	277	99

* To May 5.

From data sent by the Instituto Centrale di Statistica del Regno ,d'Italia.

Belgium

The monograph *L'encéphalite léthargique* by Van Boeckel, Bessemans and Nelis (V 19) gives a complete account of the epidemic in Belgium through 1921, including reports from the Belgian literature. Some of the original articles have not been available for the present work. The following statements, therefore, are taken freely from the monograph mentioned.

Burger and Focquet (B 431) reported a case of encephalitis lethargica seen in September, 1918, in the hospital of Beveren on the Belgian front. With this exception, the disease does not seem to have been observed in the Belgian army.

Later, Nauwelaers and Meunier (N 11) described three cases in Brussels and placed the onset of one in 1917, thus indicating the appearance of encephalitis there at about the same time as in France and England.

Early in 1919, a group of cases in Ruddervoorde attracted attention. Seventeen out of the twenty-six members of four families were taken sick, the first in February, the others in March and April.

At Harlebeke, five cases, all members of one family, developed in March and April, 1919. Other isolated cases were noted at about the same time in other localities in western Flanders, as at Aertrycke (Nélis and Baekeland) and at Blankenberghe (Retsin).

In January and February, 1920, encephalitis reappeared in about twenty villages in the same territory and also in eastern Flanders and in other provinces; cases were reported at Brussels (Laruelle), in the province of Namur (Haibe), and at Liége (Lamalle and Leroy).

During the winter of 1920-21, the disease spread from west to east in Belgium. There were numerous cases in the eastern portion. The province of Liége reported 200 affected (Lacomble-Rapport à l'Administration de l'Hygiène, Bruxelles, 1920); in Flanders, on the contrary, cases were infrequent.

Van Boeckel, Bessemans and Nelis estimate 700 cases in Belgium from the beginning of 1919 to the end of 1921. The official report for this period and the League of Nations report for the succeeding years are given in Table 1.

Table 1

Incidence of Epidemic Encephalitis by Years and Months [1919-28]

	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928*
Jan.	.....	9	175	.....	13	2	16	0	0	1
Feb.	8	11	45	.....	4	0	15	5	1	
Mar.	13	4	5	.....	2	2	6	3	1	
Apr.	3	3	4	.....	3	1	1	0	1	
May	4	6	5	.....	0	1	1	0	2	
June	.....	.....	8	.....	0	9	7	2	0	
July	2	1	.....	.....	1	2	3	0	1	
Aug.	.....	4	.....	.....	1	0	1	0	3	
Sept.	.....	5	3	.....	1	2	1	0	2	
Oct.	.....	1	3	.....	0	1	0	4	4	
Nov.	.....	1	.....	.....	2	0	2	3	1	
Dec.	3	34	.....	.....	0	2	1	2	3	
Total	33	79	248	21	27	22	54	19	19	1

* To July 14.

Of 200 cases studied by Van Boekel, Bessemans and Nelis, the morbidity and mortality by age and sex are given in Table 2.

Table 2

MORBIDITY AND MORTALITY BY AGE AND SEX IN 200 CASES [VAN BOECKEL, BESSEMANS AND NELIS]

Age group	Male		Female	
	Cases	Deaths	Cases	Deaths
Under 9 yrs.	6	0	6	0
10–20 yrs.	16	6	26	0
20–30 "	28	8	26	0
30–40 "	16	4	6	4
40–50 "	22	10	10	6
50–60 "	10	8	10	8
60–70 "	6	4	12	4
Total	104	40	96	22

The mortality rate was 31 percent.

In spite of many instances of familial affection, these authors were unable to establish one case of direct contagion

A few additional cases have been reported in the literature (Table 3).

Table 3

AGE, SEX AND SEASONAL INCIDENCE OF CASES IN THE LITERATURE

Year	Total	Sex		Age group					Quarter				
		Male	Female	1-9	10-19	20-29	30-39	N.S.	I	II	III	IV	N.S.
1919	1	.....	1	1	.....	.....	.....	.....	...	...	...	1	
1920	4	4	.....	...	1	.....	2	1	3	...	...	1	
N.S.	2	.....	2	...	.....	2	.....	.....	...	...	...	...	2
Total	7	4	3	1	1	2	2	1	3	...	...	2	2

HOLLAND

Through the courtesy of the President of the Health Council (personal communication from the Gesundheidsraad, February 29, 1928) the following information concerning the incidence of epidemic encephalitis has been obtained.

No serious epidemic of this complaint has occurred in the Netherlands. Previous to 1918, some cases were first reported as botulism. Later it was thought that these may have been cases of encephalitis lethargica. During the autumn and winter of 1918, only a few cases were described. In the beginning of 1919, there was a slight accumulation, while most cases seem to have occurred in the spring of 1920.

In January, 1924, registration of encephalitis lethargica was made compulsory. To obtain particulars concerning this complaint before that time, a questionnaire was forwarded by Prof. L. Bouman in 1924, to psychiatric and neurological

clinics, children's hospitals, asylums, etc. The results were published in 1925 (B 312). The cases reported in response to the questionnaire, in addition to those described in the Dutch literature, gave a total of 189 cases observed in the acute stage for the years 1918-23, distributed as follows:

Year	1918	1919	1920	1921	1922	1923
Children	1	4	6	10	6	8
Adults	7	11	49	39	22	26

The mortality rate was 27.5 percent.

No cases of contagion were described and there was no local accumulation of cases; these were distributed throughout the entire country.

The figures in Tables 1 and 2 showing cases and deaths of epidemic encephalitis reported for the years 1918-28, are taken from the above communication of the Gesundheidsraad and the *League of Nations Monthly Epidemiological Reports.*

Table 1

EPIDEMIC ENCEPHALITIS, CASES AND DEATHS [1918-28]

Year	Cases	Deaths
1918	8	
1919	15	
1920		69
1921		85
1922		47
1923		41

From the Gesundheidsraad and the *League of Nations Monthly Epidemiological Reports.*

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS BY YEARS AND MONTHS [1924-28]

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1924													
Cases	....	3	4	4	3	3	2	3	5	1	0	8	35
Deaths	8	2	4	12	5	2	5	1	7	2	2	2	52
1925													
Cases	11	10	35	22	9	6	5	6	7	2	6	1	120
Deaths	2	9	12	5	4	6	4	3	2	2	8	1	59
1926													
Cases	6	7	13	16	4	8	7	5	4	6	5	4	85
Deaths	7	7	9	5	5	2	5	5	5	2	3	5	59
1927													
Cases	9	2	19	16	9	7	9	7	7	6	7	3	101
Deaths	7	6	11	6	6	4	6	5	4	4	3	2	64
1928*													
Cases	....	....	....	....	....	....	....	....	....	....	....	....	34
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	24

* To May 31st.

From the Gesundheidsraad and the *League of Nations Monthly Epidemiological Reports*.

Fifty cases have been collected from the Dutch literature and tabulated in Tables 3a and 3b.

Table 3a

AGE AND SEX INCIDENCE OF 50 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age group								
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	N.S.
1917....	1	1	.....	....	.....	...	...	...	...	1	...		
1918....	1	.....	.....	1	.....	1							
1919....	13	10	3	....	.....	...	2	...	4	5	1	1	
1920....	14	13	1	....	.....	2	4	3	1	2	1	...	1
1921....	4	3	1	....	.....	...	1	...	2	1			
1922....	1	1	.....	....	.....	...	1						
1923....	2	2	.....	....	.....	1	...	1					
1924....	6	.....	5	1	1	...	1	2	...	...	...	2	
1925....	6	3	3	....	.....	2	1	1	...	...	2		
N.S.....	2	1	1	....	.....	...	1	...	1				
Total...	50	34	14	2	1	6	11	7	8	9	4	3	1

Table 3b

SEASONAL INCIDENCE OF 50 CASES REPORTED IN THE LITERATURE

Year	Quarter				
	I	II	III	IV	N.S.
1917	.....	.....	.....	.....	1
1918	.....	.....	.....	1	
1919	6	2	.....	1	4
1920	6	3	1	.....	4
1921	.....	2	.....	.....	2
1922	.....	.....	.....	.....	1
1923	1	.....	.....	1	
1924	.....	1	.....	3	2
1925	.....	3	1	1	1
N.S.	.....	.....	.....	.....	2
Total	13	11	2	7	17

GERMANY

In Germany the earliest case of lethargic encephalitis to be studied during the acute stage occurred in February, 1917. The patient, a man of thirty-eight years, presented himself at the eye clinic in Kiel, in May, and was then admitted to the medical wards of the hospital. This case, together with others which occurred in Kiel early in 1918 and in 1919, was reported by Reinhardt (R 59).

Kirschbaum (K 56a; this reference has not been available to us but is quoted by Meggendorfer, M 245), under the heading "Ueber zwei der Paralysis agitans nahestehende Krankheitsbilder," describes two cases. One of these patients

was first taken sick in Berlin at the end of May, 1917, and is thought by Meggendorfer to correspond to those in his series of chronic epidemic encephalitis.

If we accept retrospective diagnoses made from chronic conditions, we must admit the possibility of sporadic cases of epidemic encephalitis in widely separated districts in Germany in 1915, 1916 and 1917. Moewes (M 333) establishes the onset of one such case in Berlin in April, 1915; Felix Stern (S 332), in determining compensation for army service, traces two cases to the "Western front," one with onset in the fall of 1916, the other at the beginning of 1917. Meggendorfer (M 245) also describes a residual condition following acute illness "in the field" in February, 1917, and another from Berlin with onset in May of the same year. In Göttingen, one case occurred in June, 1917 (Kant: K 11); in Bonn, Meyer places the date of onset of four cases in 1917 (M 275); Kassner (K 19) lists one from Essen; and Ewald mentions one at Weiden in the winter of 1917-18.

Strümpell (Leipzig) (S 389, S 390) described his observations on twenty-five cases seen up to the date of writing in June, 1920. The age period between 15 and 25 years was chiefly affected; the three oldest were 50, 52 and 59 years. Eighteen of the number were women. In restrospect, certain cases in the hospital in 1918 were thought to have been epidemic encephalitis. Even some of the severe fatal cases seen during the preceding years and in which autopsy showed no gross anatomical changes, should probably be included in the same group, according to this author. The peak of the epidemic in Leipzig occurred during the first half of 1920.

Kayser-Petersen and Schwab (K 37) made a careful study of the epidemiology of epidemic encephalitis in Germany during the years 1918-20. They sent a questionnaire to med-

ical and psychiatric clinics and to the larger hospitals. They also made a study of the German literature available to July, 1920. Although they feel that there are many gaps in their material, it has been possible for them to show the spread of the disease during this period. They obtained reports of early cases in Dresden in October, 1916; in Leipzig in the winter of 1916-17; and in Nürnberg in 1917.

In 1918, although the total number of cases was small, reports show that they were widely scattered throughout the country; Kiel, Danzig, Berlin, Dresden, Munich, Stuttgart, Kassel, Göttingen and Dortmund being represented.

The first epidemic increase was in the winter and spring of 1919. These cases were limited for the most part to Kiel, Hamburg, Munich, Stuttgart and Tübingen. Only scattered cases appeared during the summer and fall, followed by a fresh outbreak in the winter of 1919-20. During December 1919, this was very mild in degree and was confined chiefly to the Ruhr district, but by January, 1920, encephalitis had broken out in numerous places, namely, Ruhr and Nieder-Rhein, Hamburg, Berlin, Rhein-Maingau, Nordbaden, Würtemberg, and Bavaria. The peak was reached in February and March, and the epidemic declined in April, May and June.

The above-mentioned authors have illustrated this spread of cases by maps, and are under the impression that, as far as south and middle Germany are concerned, there was a march from east to west and from north to south. It was also noticeable that in some districts, especially in middle Germany, even in the midst of affected areas, either no cases at all, or strikingly few, were reported.

An attempt to trace the epidemic appearance of encephalitis in Prussia in 1920 was also made by Fassbender (F 14). He states that the first report in Prussia was from the Gov-

ernmental division of Trier. In the subdivision Saarbrücken in the Sulzbach Valley, seventeen cases were reported from December, 1919, to the middle of January, 1920. Isolated cases were then reported in the subdivisions Trier and Wittlich. After the middle of February there was a spread of the disease to almost all the other subdivisions of Trier, although the number of cases was small. In the governmental division of Coblenz, bordering on Trier, the first cases apeared early in March, while in Aachen, several cases had been reported in the second half of January. In a number of divisions the disease appeared only sporadically. In the division Allenstein, only the little town of Rössel was visited. Not one case was reported from the divisions of Münster, Aurich, Breslau, Schneidemühl and Marienwerder. This author gives the incidence by districts with a total of 520 cases, but does not consider the figures accurate. In not one case could transmission from one person to another be traced.

Gottstein (G 114) also gives the following figures for Prussia, taken from the official reports for January to May,

Table 1

AGE AND SEX INCIDENCE OF 100 CASES [GOTTSTEIN]

Age group	Male	Female
Under 1 yr.	0	0
5–10 yrs.	3	0
10–15 "	5	3
15–20 "	12	9
20–35 "	15	13
Over 35 "	28	12
Total	63	37

1920: cases, 538; deaths, 192, or 35.6 percent. From 100 cases reported in the German literature he determined age and sex incidence as in Table 1.

Holthusen and Hapmann (H 219) reported 63 cases, which they had observed in Heidelberg during this period. The monthly incidence is given in Table 2.

Table 2

INCIDENCE BY MONTHS IN 63 CASES [HOLTHUSEN AND HAPMANN, HEIDELBURG]

Year	Month	Cases	Male	Female	Deaths recorded
1919.....	Jan.....	2	39	23	8
1920.....	Jan.....	5			
	Feb.....	16			
	Mar.....	20			
	Apr.....	13			
	May.....	5			
	June.....	2			
	Oct.....	2			

Perhaps the most intensive clinical study of encephalitis in Germany was conducted by Felix Stern in Göttingen (S 328, S 330, S 331). Although this clinic drew its cases from a relatively limited area, the author observed over four hundred in the district of Hannover, distributed by years as shown in Table 3.

Stern estimates that there must have been 60,000 cases in Germany during the epidemic, the peak occurring in 1920.

Table 3

INCIDENCE OF EPIDEMIC ENCEPHALITIS
[STERN, HANNOVER]

Year	1917	1918	1919	1920	1921	1922
Cases.....	2	7	21	232	81	83

In Bonn, Meyer (M 275) observed ninety-two cases in which the date of onset could be determined definitely. The apportionment by years is shown in Table 4.

Table 4

INCIDENCE OF EPIDEMIC ENCEPHALITIS [MEYER, BONN]

Year	1917	1918	1919	1920	1921	1922	1923*
Cases...	4	7	18	40	15	5	3

* Incomplete.

A report on the epidemic in Prussia for 1919-24 has been compiled by Deicher (D 63). He gives the incidence for the years mentioned by districts, but notes that the figures are not indicative of the true incidence, since the reporting of epidemic encephalitis is not compulsory. Each district president bases the figures on answers to questionnaires, which are often incomplete. The author believes that the correct figures would probably be double or triple those given here. Table 5 has been prepared from the tables in the text in order to show the incidence by years for the twenty-four districts that so reported. Ten districts reported for the six-year period as a whole and not by individual years. The total number of

cases thus obtained does not agree with the author's total, 11,317, owing to a discrepancy of eight between the total as given in the text for Frankfurt a. O. and the total obtained by adding together the figures given for each of the six years in that district. There was no report on deaths from six districts. The mortality rate was 15 percent; 32 percent of the cases became chronic. The peak of the epidemic was in the years 1923 and 1924. In 1925, there were only 217 cases in all Prussia.

Table 5

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN PRUSSIA
[1919-24, DEICHER]

Years	Cases	Deaths
1919	541	68
1920	1,448	176
1921	1,358	136
1922	1,596	137
1923	1,861	141
1924	2,187	137
1919–1924*	2,318	903
Total	11,309	1,698

* 10 districts.

The age was not always stated, but the incidence was apparently greatest in the age-group 31-60 years.

No difference between city and country could be determined with respect to morbidity.

Eight instances of possible contact infection, involving twenty-seven people, are described. This small number of

Table 6

Deaths and Mortality from Encephalitis Lethargica Notified in German Towns of 15,000 Inhabitants or More During the Years 1925-28*

Month	1925		1926		1927		1928	
	Actual Figures	Per 10,000†	Actual Figures	Per 10,000	Actual Figures	Per 10,000	Actual Figures	Per 10,000
Jan.	33	0.1	40	0.2	29	0.1	16	0.1
Feb.	22	0.1	30	0.1	16	0.1	7	0.03
Mar.	17	0.1	17	0.1	20	0.1	19	0.1
Apr.	9	0.04	16	0.1	5	0.02	20	0.1
May	6	0.03	18	0.1	4	0.02		
June	11	0.1	16	0.1	7	0.03		
July	7	0.03	12	0.1	8	0.03		
Aug.	6	0.03	23	0.1	14	0.1		
Sept.	6	0.03	16	0.1	7	0.03		
Oct.	4	0.02	11	0.05	8	0.03		
Nov.	12	0.1	12	0.1	12	0.1		
Dec.	11	0.1	13	0.1	14	0.1		
Total‡	144	0.1	224	0.1	144	0.1		
Definitive result according to the annual report	142	0.1	217	0.1	Not yet available			

* Population, Jan., 1925: 26,535,000; April, 1928: 28,057,000.

† Rate per 10,000 inhabitants (monthly average of population) calculated on an annual basis.

‡ Provisional figures.

From a report sent to the Survey through the courtesy of the Reichsgesundheitsamt.

probable direct transmissions shows that the infectiousness of the disease cannot be very great.

Epidemic encephalitis has not yet been made reportable in Germany, but Table 6 has been taken from a report sent to the Survey through the courtesy of the Reichsgesundheitsamt.

Nine hundred and eighty-six cases reported in the German literature have been tabulated according to sex, age, and seasonal incidence. (Tables 7a and 7b).

Table 7a

AGE AND SEX INCIDENCE OF 986 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age group									
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.
1915	1	.....	1	....	.....	...	...	...	1					
1916	1	1	.....	....	.....	...	...	1						
1917	12	5	3	4	.....	...	1	2	2	2	...	...	...	5
1918	42	26	14	2	.....	3	5	10	8	2	8	2	...	4
1919	164	116	41	7	1	7	41	32	36	28	13	3	...	3
1920	448	271	171	6	1	45	130	107	93	46	16	4	1	5
1921	91	55	35	1	1	6	25	15	19	19	2	2	1	1
1922	46	28	18	....	.....	2	10	12	12	4	4	2		
1923	70	38	27	5	.....	7	11	22	15	8	5	1	...	1
1924	19	5	13	1	.....	5	1	6	2	1	...	1	...	3
1925	7	3	4	....	.....	2	2	1	...	1	1			
1926	5	3	2	....	.....	...	3	2						
1927	3	1	2	....	.....	...	...	1	1	1				
N.S.	77	43	33	1	.....	1	23	17	21	5	6	...	...	4
Total	986	595	364	27	3	78	252	228	210	117	55	15	2	26

Table 7b

SEASONAL INCIDENCE OF 986 CASES REPORTED IN THE LITERATURE

Year	Quarter				
	I	II	III	IV	N.S.
1915	.....	1			
1916	.....	.....	.....	1	
1917	4	5	.....	.....	3
1918	6	6	3	11	16
1919	91	23	7	26	17
1920	245	85	17	29	72
1921	48	14	9	8	12
1922	13	3	6	19	5
1923	40	9	8	4	9
1924	11	6	.....	2	
1925	2	.....	.....	5	
1926	1	1	3		
1927	.....	2	.....	.....	1
N.S.	.....	.....	.....	.....	77
Total	461	155	53	105	212

AUSTRIA

It has been established that cases of the disease now known as epidemic encephalitis had been observed and described in Rumania and in France previous to those reported from Austria. To von Economo, however, belongs the credit of having brought this disease-entity into prominence and of having called it to the attention of physicians, under the name of encephalitis lethargica (E 11a). Economo's first description included seven acute cases which occurred

in Vienna in the period, January to March, 1917. In other articles (E 11, E 13) six additional cases, with onset January to May, 1917, were reported.

On February 16, 1917, a cadet was admitted to the Marine Hospital in Pola in the province of Istria (now a part of Italy). The clinical history of the case is described in full by Rozankowski (R 289). The author believes that this case belongs in the same group as those described by von Economo. He calls attention to the fact that the patient had been on leave in Baden, near Vienna, and in Vienna, from February 3 to 12, just at the time that von Economo's cases were observed in Vienna.

Hoff (H 189) gives a very full report of the cases of epidemic encephalitis observed at the Psychiatric and Neurological Clinic (Economo's) in Vienna, from January, 1916, to April 30, 1923. He lists thirteen cases in November, 1916, and one in December; seven of the November and four of the December cases were observed at that time during the acute stage; for the others the date of onset was determined from the history given. Moreover, the author notes that the number of cases reported as meningitis increased noticeably in Vienna at this time, and believes that some of these were undoubtedly epidemic encephalitis.

A study of incapacitated soldiers with sequelae following encephalitis lethargica, was made by Stiefler, of Linz (S 364) and showed that in one case the acute illness had occurred in March, 1915; another soldier, placed in a Rumanian prison in December, 1916, developed encephalitis in February, 1917. The author states that a case of encephalitis lethargica was diagnosed at the Innsbruck Neurological Clinic in January, 1917, and that the onset of another definite case occurred in March, 1917, in Trient.

A series of 160 cases, occurring in Vienna early in 1920, was reported by Redlich (R 40) by age and sex as shown in Table 1.

Table 1

AGE AND SEX INCIDENCE OF 160 CASES [REDLICH, VIENNA]

Age group	Cases	Percentage	Sex	Cases	Percentage
1–10	3	1.8	Male	109	68
10–20	45	28.1	Female	51	32
20–30	27	16.8			
30–40	38	23.7			
40–50	31	19.3			
50–60	13	8.1			
Over 60	3	1.8			

Table 2

AGE, SEX AND SEASONAL INCIDENCE OF 59 CASES [KURZ-GOLDENSTEIN, LINZ]

Month	Cases	Age-group	Cases	Per-centage	Sex	Cases
Jan.	11	1–10	3	5.08	Male	34
Feb.	29	10–20	15	25.42	Female	25
Mar.	17	20–30	13	22.03		
Apr.	1	30–40	10	16.94		
May	1	40–50	10	16.94		
.....	.....	50–60	5	8.47		
.....	.....	60–70	2	3.33		
.....	.....	70–80	1	1.69		
Total	59	.....	59	.....	.....	59

Stiefler and Kurz-Goldenstein (S 366) studied a smaller group of 59 cases in Linz and environs in 1921.

Epidemic encephalitis was not made reportable in Austria until January, 1927, so that figures for the country as a whole are not available. Hoff's study of the cases registered at the University Clinic in Vienna may serve to show the trend up to 1923. The figures given here in Tables 3, 4 and 5 include both the cases that were seen in the acute stage and those in which the date of onset was established from the history, even though the patients were first observed when the condition had become chronic.

Table 3

SEASONAL INCIDENCE OF EPIDEMIC ENCEPHALITIS
[1916-23, HOFF, VIENNA]

	1916	1917	1918	1919	1920	1921	1922	1923*	Total
Jan.	.....	3	1	11	43	13	11		
Feb.	.....	1	.....	8	477	10	14	3	
Mar.	.....	1	.....	16	200	26	9	19	
Apr.	.....	2	1	3	39	4	10	26	
May	.....	1	.....	2	6	.....	5		
June	.....	1	.....	.....	3				
July	.....	.....	2	.....	.....	.....	3		
Aug.	.....	1	2	.....	1	2	4		
Sept.	.....	2	1	2	1	1	1		
Oct.	.....	.....	52						
Nov.	13	1	64	2	.....	.....	2		
Dec.	8	1	51	1	6	.....	1		
Total	21	14	174	45	776	56	60	48	1,194

* To Apr. 30.

Table 4

AGE AND SEX INCIDENCE OF 1,156 CASES [HOFF, VIENNA]

Age Group	Cases	Sex	Cases
Up to 5 yrs.	17	Male	642
5–10 yrs.	52	Female	471
10–15 "	123	N.S.	43
15–20 "	183		
20–25 "	242		
25–30 "	202		
30–35 "	139		
35–40 "	74		
40–45 "	83		
45–50 "	19		
50–55 "	10		
55–60 "	9		
60–65 "	3		
Total	1,156		1,156

It must be taken into consideration that only a certain stratum of society visits the clinic and that, moreover, we are dealing with a city population. With these points in mind, Hoff concludes that professional men and skilled laborers were noticeably affected, and of these, metal workers particularly so. Even though the metal industry in Austria had expanded considerably, the author feels that the number of these workers affected is relatively high.

No evidence of a focal point for the infection was found: "The cases were scattered over the whole of Vienna like rain." In spite of the mass of material in the clinic, there was not one case of definite contact-infection established.

Table 5

CLASSIFICATION ACCORDING TO OCCUPATION (MALES)
[HOFF, VIENNA]

Professions	28
Teachers, lawyers, physicians, students, etc.	
Officials	34
Manual workers	31
Tailors, joiners, cobblers, etc.	
Metal-workers	133
Smiths, puddlers, mechanics	
Business men	56
Chauffeurs, railroad conductors, etc.	7
Peasants (farmers)	51
Foresters	10
Clerks	7
Carpenters, etc.	14
Laborers	87
Others	33

Of 265 acute cases admitted to the hospital, 77 (29 percent) died.

One thousand three hundred and ninety-three cases reported in the literature of Austria (including some of Hoff's cases) have been tabulated in Tables 6a and 6b.

Table 6a

AGE AND SEX INCIDENCE OF 1,393 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age group								
		Male	Female	N.S.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.
1915	1	1	.....	.....	1								
1916	21	10	8	3	2	3	4	2	1	...	...	...	10
1917	28	10	10	8	...	5	6	6	3	1	...	1	6
1918	179	91	79	9	4	25	27	20	11	3	1	...	88
1919	49	25	22	2	2	19	16	8	3	...	1		
1920	906	487	348	71	22	133	170	143	90	24	6	...	318
1921	76	37	33	6	1	2	3	7	5	1	1	...	56
1922	68	31	30	7	...	4	...	1	1	2	...	...	60
1923	52	30	20	2	...	1	1	...	1	...	1	...	48
1924	2	1	1	.....	...	...	2						
1925	3	2	1	.....	...	1	1	1					
1926	1	.....	1	.....	...	...	...	...	1				
N.S.	7	5	2	.....	...	2	3	2					
Total	1,393	730	555	108	32	195	233	190	106	31	10	1	586

Table 6b

SEASONAL INCIDENCE OF 1,393 CASES REPORTED IN THE LITERATURE

Year	Quarter				
	I	II	III	IV	N.S.
1915	1				
1916				21	
1917	17	6	3	2	
1918	1	2	5	170	1
1919	37	5	2	5	
1920	829	61	2	9	5
1921	61	6	4		5
1922	40	16	8	3	1
1923	24	27			1
1924				1	1
1925	1	1	1		
1926	1				
N.S.					7
Total	1,012	124	25	211	21

Hungary

A few cases from Hungary have been reported in the literature and are analysed in the accompanying table.

Age, Sex and Seasonal Incidence of 24 Cases Reported in the Literature

Year	Total	Sex		Age group							Quarter				
		Male	Female	1–9	10–19	20–29	30–39	40–49	50–59	N.S.	I	II	III	IV	N.S.
1919	1	1	.....	...	...	...	1	...	...	....	...	...	...	...	1
1920	19	12	7	1	5	6	3	2	1	1	18	...	...	...	1
1921	1	.....	1	...	...	1	...	...	...	....	1	...			
1924	2	2	.....	...	...	2	...	...	...	.....	...	2			
N.S.	1	.....	1	...	...	1	...	...	...	.....	...	...	...	...	1
Total	24	15	9	1	5	10	4	2	1	1	19	2	...	...	3

Denmark

Madsen (M 25) published the figures for cases and deaths from epidemic encephalitis in Denmark to the close of 1920, and gives the incidence and mortality by age and sex in Table 1.

Table 1

AGE AND SEX DISTRIBUTION OF 133 CASES AND 55 DEATHS [MADSEN]

Age group	Cases			Deaths		
	M	F	Total	M	F	Total
Under 5 yrs.	3	4	7	1	3	4
5–10 yrs.	4	3	7	5	3	8
10–15 "	12	8	20	2	3	5
15–20 "	19	12	31	5	6	11
20–25 "	17	7	24	8	0	8
25–35 "	17	17	34	9	11	20
35–45 "	23	4	27	8	4	12
45–55 "	16	12	28	6	4	10
55–65 "	15	4	19	5	3	8
65–75 "	3	3	6	3	3	6
75–85 "	1	1	2	0	1	1
Age not stated	3	1	4	1	0	1
Total	133	76	209	55	41	96

The mortality rate was 45.9 percent.

The officially reported cases of epidemic encephalitis in Copenhagen are given by Neel (N 34) by years (Table 2).

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN COPENHAGEN [NEEL]

Year	1919	1920	1921	1922	1923	1924	1925	1926*
Cases	2	25	4	11	12	17	16	5

* To July 1.

The figures in Table 3 for cases and deaths in Denmark, 1919-28, have been taken from the *League of Nations Monthly Epidemiological Reports.*

Table 3

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN DENMARK BY YEARS AND MONTHS [1919-28]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1919													
Cases							1	3	0	0	5	9	26
1920													
Cases	22	10	13	17	40	25	25	18	13	7	5	1	226
1921													
Cases	23	39											140
Deaths													57
1922													
Cases		4		4	4	1	6	2	1	0	2	3	42
Deaths													39
1923													
Cases	12	23	21	7	5	2	5	3	2	6	3	2	91
Deaths													30
1924													
Cases	3	4	10	14	17	8	6	7	5	6	10	15	105
Deaths													57
1925													
Cases	19	22	23	18	8	4	10	10	11	8	9	5	150
Deaths													76
1926													
Cases	7	2	8	5	10	5	1	8	5	9	5	5	72
Deaths													65
1927													
Cases	12	16	16	10	7	18	5	8	4	6	10	4	116
1928*													
Cases													67

*To June 30.

From the *League of Nations Monthly Epidemiological Reports.*

Only 10 cases have been collected from literature (Table 4).

Table 4

AGE, SEX AND SEASONAL INCIDENCE OF 10 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group				Quarter				
		M	F	10–19	20–29	30–39	40–49	I	II	III	IV	N.S.
1918	1	.....	1	.....	1	.....	.....	....	....	....	....	1
1920	4	1	3	1	1	1	1	....	1	....	....	3
1922	1	.....	1	.....	.....	1	.....	....	....	....	....	1
1923	1	1	.....	.....	1	.....	.....	....	....	....	....	1
1924	1	1	.....	.....	1	.....	.....	....	....	....	....	1
1925	1	.....	1	1	.....	.....	.....	....	1			
N.S.	1	.....	1	1								
Total	10	3	7	3	4	2	1	....	2	....	....	7

NORWAY AND SWEDEN

Sporadic cases of epidemic encephalitis were noted in Sweden in October and November, 1918 (M 72). The onset of other cases is placed retrospectively in January and February, 1919; but cases did not appear in groups in Norway or Sweden until the summer and fall of 1919.

Two local epidemics in the winter of 1920-21 have been studied intensively. Kristensen (K 146) reported an epidemic beginning in November, 1920, in the Royal Upland Infantry Regiment and in a detachment of Royal Scandinavian Hussars. There were four pronounced and nineteen abortive cases, ages sixteen to twenty-two years, all male. The onset of the first two cases was on November 28 and 29 respective-

ly. These two men belonged to different regiments; neither had been on leave for some time previous, nor had had visitors. Although both visited the canteen, they probably did not know each other. No direct transmission could be traced in any instance. The author considers the possibility of transmission by bacilli-carriers and believes the incubation period to be from eight to eighteen days. The incidence of encephalitis in the five counties in the neighborhood of the garrison is given as follows:

SEASONAL INCIDENCE [KRISTENSEN, SWEDEN]

1920			1921	
Oct.	Nov.	Dec.	Jan.	Feb.
3	10	31	212	87

Kling and Liljenquist (K 81) and (K 66) describe an epedemic in Vilhelmina, southern Lappland, occurring in February, 1921. The morbidity varied from 7.1 percent to 45 percent. All the inhabitants of four small hamlets were examined. In some families, several members contracted the disease simultaneously; in two houses almost all the members were affected. There were many light cases. These authors found nothing to justify the belief that epedemic encephalitis can be transmitted by water or milk, or by bugs, fleas or lice (there are no other insects in Sweden in the winter). Dogs and cats when examined, showed no evidence of the disease. The period of incubation was thought to be ten days. There was such a rapid spread of the disease over a wide area in two months that it could scarcely have been due to human contact.

Table 1

Epidemic Encephalitis, Cases and Deaths in Norway [1919-1928]*

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1919													
Cases....	1	1	1	....	....	....	....	....	....		18		21
1920													
Cases....		10		....	....	....	....	....	....	....	....	....	10
1921													
Cases....	....	....	....	....	....	....	....	....	....	....	....	....	55
1922													
Cases....	....	....	2	1	0	0	0	0	0	1	2	0	7
1923													
Cases....	3	4	0	3	0	0	0	0	0	0	0	0	10
1924													
Cases....	0	4	5	0	0	0	0	0	0	1	0	0	10
Deaths..	0	0	1	0	0	0	0	0	0	0	1	0	2
1925													
Cases....	2	4	3	0	1	0	1	1	2	0	0	0	14
Deaths..	0	1	1	0	0	0	1	0	0	0	0	0	3
1926													
Cases....	0	2	1	0	0	12	0	0	0	0	0	0	15
Deaths..	0	0	1	0	0	0	0	0	0	0	0	0	1
1927													
Cases....	0	0	0	1	0	2	0	0	1	0	0	1	5
1928†													
Cases....	....	....	....	....	....	....	....	....	....	....	....	....	8

* Official reports are for "cities" only.

† To May 31.

From the *Public Health Reports* and the *League of Nations Monthly Epidemiological Reports.*

Table 2

EPIDEMIC ENCEPHALITIS, CASES AND DEATHS IN SWEDEN [1919-1928]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1919													
Cases	....	....	....	....	....	....	....	....	....	....	....	....	5
1920													
Cases	....	....	....	....	....	....	13	2	....	6	34	75	352
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	134
1921													
Cases	....	978	....	....	....	....	....	....	....	....	....	....	1,512
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	370
1922													
Cases	....	....	....	....	....	....	....	5	3	....	19	45	192
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	79
1923													
Cases	102	154	93	39	22	19	25	17	16	16	13	20	536
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	181
1924													
Cases	32	63	43	25	19	11	14	22	21	20	13	17	301
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	119
1925													
Cases	14	17	22	15	8	14	17	17	23	16	7	16	198
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	106
1926													
Cases	12	13	20	15	12	13	13	11	6	16	10	12	153
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	96
1927													
Cases	10	10	8	6	12	15	18	6	10	9	11	14	129
1928*													
Cases	....	....	....	....	....	....	....	....	....	....	....	....	85

* To June 30.

From the *Public Health Reports* and the *League of Nations Monthly Epidemiological Reports.*

The figures in Tables 1 and 2 showing cases and deaths in Norway and Sweden for the year 1919-28 have been taken from the *Public Health Reports* (74) and from the *League of Nations Monthly Epidemiological Reports.* In Sweden, epidemic encephalitis was made reportable July 1, 1920.

Eighty-one cases from Norway and Sweden, reported in the literature, are analysed in Table 3.

Table 3

AGE, SEX AND SEASONAL INCIDENCE OF 81 CASES REPORTED IN THE LITERATURE, NORWAY AND SWEDEN

Year	Total	Sex			Age group									Quarter			
		Male	Female	N.S.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.	I	II	III	IV
1918	2	...	2	...	...	...	1	1	...	...	...	...	...	...	...	...	2
1919	16	11	5	...	...	2	7	3	...	2	...	2	...	2	1	9	4
1920	22	20	1	1	...	3	12	2	2	1	...	...	2	2	6	1	13
1921	29	14	12	3	2	8	13	...	4	...	2	...	...	24	4	...	1
1922	1	1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
1923	5	3	2	...	...	...	1	3	1	...	...	...	...	2	2	1	
1924	2	...	2	...	...	...	1	...	1	...	...	...	...	2			
1925	3	...	3	...	...	1	...	1	1	...	...	...	...	3			
N.S.	1	1	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1
Total	81	50	27	4	2	14	36	10	10	3	2	2	2	35	13	11	22

Finland

The incidence of epidemic encephalitis in Finland as given in the *League of Nations Monthly Epidemiological Reports* is shown in Table 1.

Table 1

Incidence of Epidemic Encephalitis in Finland

	1920	1921	1922	1923	1924	1925	1926	1927	1928*
Jan.	.....	.....	.....	22	3	6	2	3	
Feb.	.....	.....	16	29	5	6	1	0	
Mar.	.....	.....	5	12	6	5	3	0	
Apr.	.....	.....	4	6	6	0	0	1	
May	.....	.....	2	1	3	0	2	0	
June	.....	.....	.....	1	3	7	2	1	
July	.....	.....	.....	1	1	0	1	0	
Aug.	.....	.....	.....	2	2	1	0	0	
Sept.	.....	.....	1	1	2	0	1	0	
Oct.	.....	.....	1	2	2	3	0	0	
Nov.	.....	.....	1	2	2	1	1	0	
Dec.	.....	.....	1	4	8	1	0	1	
Total	239	1,095	46	83	43	31	13	6	3

* To June 15.

From the *League of Nations Monthly Epidemiological Reports.*

Twenty-three cases reported in the Finnish literature have been analysed in Table 2.

Table 2

AGE, SEX AND SEASONAL INCIDENCE OF 23 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group					Quarter				
		Male	Female	1–9	10–19	20–29	30–39	40–49	I	II	III	IV	N.S.
1920	9	5	4	1	7	1	.....	.....	...	...	...	9	
1921	14	6	8	1	3	4	2	4	12	...	...	...	2
Total	23	11	12	2	10	5	2	4	12	...	...	9	2

In addition, Hagelstan (H 8) reported ninety cases (mortality 16 percent) observed in 1920-21 in the Maria Hospital in Helsingfors.

POLAND

The first known case of epidemic encephalitis in Poland occurred in February, 1917, and was presented at that time by Sterling before the section of Neurology and Psychiatry of the Medical Society of Warsaw (S 318). Sterling states that several other cases, most of them in Warsaw, occurred during the early months of the same year.

Bychowski (B 481), writing in 1921, believes on looking back that there were cases of epidemic encephalitis in Warsaw in 1918 and 1919, although the disease did not appear in epidemic form until the end of December, 1919. During the period from January to March, 1920, there were several

hundred cases in Warsaw, according to this author's estimate. He himself saw forty cases in the acute stage, and later added to that number twenty who presented residual conditions.

In March, 1920, epidemic encephalitis was made reportable in Poland. The figures for the great epidemic in 1920 are given by Sterling (S 318) (Table 1). The author calls attention to the fact that the peak was reached in April, May and June, instead of in the winter months, as so frequently reported. The mortality rate was 19.3 percent.

Table 1

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN POLAND [1920, STERLING]

1920	Cases	Age Group	Cases	Male		Female	
				Cases	Deaths	Cases	Deaths
		Years					
Jan.	.....	1–4	28				
Feb.	.....	5–14	232				
Mar.	104	15–19	315				
Apr.	730	20–29	306				
May	219	30–39	244				
June	243	40–49	130				
July	83	50–59	83				
Aug.	51	60–69	28				
Sept.	22	70+	8				
Oct.	2						
Nov.	8						
Dec.							
Total	1,462	.....	1,374	723	128	651	133

Neither social nor economic conditions, nor the influence of city or country, played a rôle in the development or in the gravity of the disease; patients representing thirty-four occupations are listed, among them that of laborer, official, bank officer, soldier, engineer, student, chauffeur, servant, etc. The proportion of the Jewish race affected seemed a little high, 526 as against 848 Gentiles, whereas only about 14 percent of the population was Jewish.

Six instances of possible contagion are mentioned.

Observations on 126 cases in Warsaw during this epidemic have been reported by Goldflam (G 89). Part of these were seen in association with other physicians and may therefore appear in other statistics. The month of onset was known for 102 cases. Goldflam's figures have been grouped in Table 2.

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN WARSAW [1920, GOLDFLAM]

1920	Cases	Deaths	Age group	Cases	Male	Female	Deaths	Male	Female	Mortality rate Percentage
Jan.	16	....	Under 10 yrs.	8	7	1				
Feb.	56	· 7	to 20 "	46	31	15				
Mar.	25	5	" 30 "	41	25	16				
Apr.	4	1	" 40 "	17	11	6				
May	1	....	" 50 "	7	6	1				
			" 60 "	6	5	1				
			Over 60 "	1	....	1				
Total	102	13		126	85	41	14	11	· 3	11.3

Direct evidence of contagion was not obtained; however three instances are described in which there was a possible transmission from one patient to another.

In a second article Sterling (S 319) gives an account of events in Poland after 1920. Sporadic cases occurred during 1921. In 1922 there was a new outbreak, and it was noted that it began in the month of August. Shortly before the first cases occurred in Poland, there were reports of the appearance of the disease in Alsace, Lorraine and Westphalia. Most of

Table 3

Incidence of Epidemic Encephalitis in Poland [1922-23, Sterling]

1922	Men	Women	Infants	Total
May	15	3	0	18
June	17	12	2	31
July	4	8	0	12
Aug.	46	32	3	81
Sept.	59	44	5	108
Oct.	23	20	2	45
Nov.	26	25	3	54
Dec.	32	25	1	58
Total	222	169	16	407
1923				
Jan.	37	37	1	75
Feb.	68	54	4	126
Mar.	38	35	1	74
Apr.	20	17	0	37
Total	163	143	6	312

the cases occurred in the northwest part of Poland, and this new epidemic seemed to have been a little delayed in other parts of the country. This would indicate, Sterling believes, that the epidemic traveled from west to east in Poland. No further determinations on the contagiousness of encephalitis were made, although one instance, observed at Warsaw, showed a probable transmission from a mother to nursling and to a servant.

The figures for the incidence in 1922 and 1923 are given in Table 3.

In addition to the groups described above, only twenty-seven cases have been collected from the Polish literature. These are tabulated in Table 4.

Table 4

AGE, SEX AND SEASONAL INCIDENCE OF 27 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group						Quarter				
		Male	Female	1–9	10–19	20–29	30–39	40–49	N.S.	I	II	III	IV	N.S.
1917	1	1	.....	...	1	...	...	...	....	1				
1918	1	.....	1	...	1	...	...	...	....	....	....	....	....	1
1919	1	1	.....	...	1	...	...	...	....	....	....	....	....	1
1920	18	10	8	4	4	5	1	3	1	16	1	....	....	1
1921	2	1	1	...	1	...	...	1	....	1	....	....	1	
1922	1	.....	1	...	1	...	...	...	....	....	....	1		
1924	2	1	1	1	...	1	...	...	....	....	1	1		
N.S.	1	1	.....	...	...	...	...	1	....	....	....	....	....	1
Total	27	15	12	5	9	6	1	5	1	18	2	2	1	4

Czechoslovakia

In April, 1917, a woman from Kladno, near Prague, was admitted to a medical clinic in Prague. Pribram (P 214) has given the history of the case in detail; he believes that he established the similarity between this condition and that described by von Economo, and that this was a sporadic case of encephalitis lethargica.

Later, Herrmann and Wotke (H 150), writing in 1925, observed a case of Parkinsonism in a man then twenty-five years of age, and made a retrospective diagnosis of epidemic encephalitis, with onset in 1917. The patient had been serving in the army at that time.

Jaksch-Wartenhorst (J 22) has reported the cases that came under his observation in Prague. He saw 36 acute cases distributed as shown in Table 1. Seven of these were fatal (3 Male, 4 female). The highest morbidity was in the age period from 16-20 years. Up to December 31, 1922, this author saw 100 chronic cases (55 male, 45 female).

Table 1

Incidence by Months of 36 Cases [Jaksch-Wartenhorst, Prague]

Month	1920 Cases	1921 Cases	1922 Cases	1923 Cases	Total Cases
Jan.	5	1	.....	2	8
Feb.	12	0	.....	4	16
Mar.	5	0	.....	4	9
Apr.	2	0	.....	0	2
June	1	0	.....	0	1
Total	25	1	0	10	36

The cases of epidemic encephalitis observed at the Czech Medical Clinic in Prague from 1919 to 1928 have been reviewed by Syllaba and Henner (S 401a) who believe that the first sporadic cases in the country occurred in 1918, but escaped attention until studied for chronic ailments. The epidemic began in 1919 but did not assume prominence until January, 1920. Statistics for the country are not complete, but the authors estimate at least 1 case to every 1,000 population for the nine-year period, with a total of 10,000 or more cases.

In all, about 1,000 patients were seen at the first Czech Medical Clinic. The greatest number of cases developed in the winter months. In no instance did the acute onset occur in June, July or August. The cases were about evenly divided between the two sexes.

The greatest age incidence was in the period between six

Table 2

AGE, SEX AND SEASONAL INCIDENCE OF 39 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group					Quarter				
		Male	Female	10–19	20–29	30–39	40–59	N.S.	I	II	III	IV	N.S.
1917	2	1	1	1	1	.....	.....	....	..	1	...	...	1
1919	1	1	.....	.....	1	.....	.....	....	..	..	...	...	1
1920	28	11	17	3	11	9	3	2	23	3	...	...	2
1921	6	3	3	3	1	.....	1	1	4	..	1	1	
1923	1	1	.....	.....	.....	.....	1	....	1				
1925	1	1	.....	.....	1	.....	.....	....	1				
Total	39	18	21	7	15	9	5	3	29	4	1	1	4

Table 3

Incidence of Epidemic Encephalitis in Czechoslovakia by Years [1922-28]

Year	1922	1923	1924	1925	1926	1927	1928*
Cases.....	150	366	97	189	54	70	
Deaths.....	.....	116	33	43	21	150	34

* To June 30.

From the *League of Nations Monthly Epidemiological Reports.*

Table 4

Incidence of Epidemic Encephalitis in Czechoslovakia by Months [1923-1927]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1923													
Cases....	70	100	85	44	14	18	6	5	3	6	4	11	366
Deaths..	23	27	18	15	6	12	0	1	3	3	3	5	116
1924													
Cases....	7	9	22	25	8	5	8	0	3	0	6	4	97
Deaths..	2	1	9	6	1	2	4	0	2	0	4	2	33
1925													
Cases....	14	25	40	31	10	14	14	8	3	10	2	2	189
Deaths..	1	1	8	10	4	1	9	3	1	2	2	1	43
1926													
Cases....	4	10	6	6	4	8	5	2	3	2	1	2	54
Deaths..	3	1	2	3	2	4	0	1	1	1	1	2	21
1927													
Cases....	32	13	10	3	1	1	3	0	3	2	0	2	70
Deaths..	61			30			33			26			150

From the *League of Nations Monthly Epidemiological Reports.*

and thirty-six years. No patients were over sixty years of age. In only two instances was there any possibility of contagion; the first was that of a married couple, the second that of a physician who treated another physician suffering from encephalitis.

Epidemics of greater or less severity occurred each winter from 1919 to 1925. No new cases were seen between October, 1925 and October, 1927. Each epidemic was preceded by a short outbreak of epidemic hiccup.

Thirty-nine additional cases from Czechoslovakia, reported in the literature, are tabulated in Table 2.

The figures in Tables 3 and 4 have been taken from the *League of Nations Monthly Epidemiological Reports* and show the incidence in Czechoslovakia by years for the period 1922-28 and the incidence by months for the period 1923-27.

Rumania

Apparently the first case of encephalitis to be reported during this epidemic was observed at the Marcutza Hospital, Bucarest, Rumania, May, 1915. An account of this case was published in 1916 by Obregia, Urechia and Carniol (O 6a) under the title "Encéphalite hémorragique avec un diplocoque encapsulé."

Urechia (U 7) makes the following statement. "In reviewing a study of the histories on the service of M. le Professeur A. Obregia, I found in the month of April a case of the myoclonic type and another case of the lethargic type with an acute course in the month of May, 1915. We published the second case in the review Spitalul together with MM. Obregia et Carinol. We pointed out the perivascular infiltration and predominance of the lesions along the ventricles. In the

illustration which accompanied our article we indicated the cellular inclusions which I described that year in a note addressed to the Société de Biologie. On the same service of M. Obregia, I made, during the years 1915-16, three other autopsies of hemorrhagic encephalitis which, according to the examination made again this year prove to be also those of epidemic encephalitis."

The two cases above-mentioned and eight others, all with autopsies, are described in the article quoted. In all, 89 Rumanian cases have been collected from the literature (Table 1).

Table 1

AGE, SEX AND SEASONAL INCIDENCE OF 89 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group						Quarter				
		Male	Female	10–19	20–29	30–39	40–49	70+	N.S.	I	II	III	IV	N.S.
1915*	2	1	1	...	...	2	...	...	...	...	2			
1918	1	1	.....	...	...	...	1	...	...	...	1			
1919	3	2	1	2	...	...	...	...	1	...	...	...	3	
1920	61	31	30	18	22	7	6	1	7	21	12	24	2	2
1921	12	9	3	1	6	2	2	1	....	7	2	1	...	2
1922	5	3	2	1	3	1	...	...	....	2	...	1	...	2
1923	1	1	.....	...	1	...	...	...	.....	...	...	...	1	
1924	1	1	.....	...	1	...	...	...	....	1				
1925	1	.....	1	...	...	1	...	...	....	...	...	...	...	1
N.S.	2	.....	2	1	1	...	...	...	....	...	...	...	...	2
Total	89	49	40	23	34	13	9	2	8	31	17	26	6	9

*Urechia: *Arch. internat. de neurol* 2: 65, 1921: Ten case histories given, some traced back to epidemic of 1915. *Compt. rend. Soc. de biol.* 85: 581, 1921: In May, 1915, author reported the first case of encephalitis in Bucarest.

The figures in Table 2 for the incidence of epidemic encephalitis in Rumania have been taken from the *League of Nations Monthly Epidemiological Reports.*

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN RUMANIA BY MONTHS [1925-1928]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1925													
Cases	....	....	20	2	4	1	2	4	3	0	0	0	36
Deaths	....	....	6	3	1	1	1	....	....	....	....	0	12
1926													
Cases	0	1	1	1	1	2	0	3	0	0	0	0	9
Deaths	0	0	0	0	0	1	0	0	0	0	0	0	1
					9								
1927													
Cases	0	0	0	3	2	0	0	1	0	1	4	1	16
Deaths	0	0	0	....	....	....	....	....	....	....	....	....	2
			2										
1928†													
Cases	2	1	....	....	....	....	....	....	....	....	....	....	7

† Up to June 16.

From the *League of Nations Monthly Epidemiological Reports.*

BULGARIA

Only a few cases from Bulgaria have been reported in the literature, as indicated in the accompanying table.

AGE, SEX AND SEASONAL INCIDENCE OF 19 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group				Quarter				
		Male	Female	10–19	20–29	30–39	40–49	I	II	III	IV	N.S.
1919	1	.....	1	.....	1	.....	.....	1				
1920	14	14	.....	3	6	2	3	6	3	...	1	4
1923	2	1	1	.....	1	1	.....	...	...	...	1	1
1924	2	1	1	.....	1	1	.....	...	1	...	1	
Total	19	16	3	3	9	4	3	7	4	...	3	5

The *League of Nations Monthly Epidemiological Reports* give the following figures for 1924-27. 1924: 146 cases were treated in the hospitals, mostly during the latter half of the year. The disease was not previously notifiable. 1925: cases, 67; mortality, 13 percent. 1926: cases, 12; deaths, 6. 1927: cases, 9; deaths, 6.

RUSSIA

According to Pappenheim (P 28), cases of encephalitis lethargica were observed in Russia, first in the Ukraine in the fall of 1919, then in central Russia in the fall of 1920.

The first publication from the Ukraine, Pappenheim says, was by J. Raimist in Odessa. Raimist reported 52 cases observed in the period from November 23, 1919 to January 9, 1920 (with 40 additional cases by March 17). Of these, 23 were males, 29 females; 42 percent came within the age period 20-40 years; the mortality was 17.3 percent.

Forty-six cases were reported by Heimanowitsch in Charkow (also in the Ukraine), from the middle of November, 1919 to the end of February, 1920. Eight (18 percent) were fatal.

Fleischmann's observations in Kiew (in the Ukraine) are also quoted by Pappenheim, but no figures are given as to the number of cases.

In central Russia, where encephalitis first appeared a year later, that is, in the fall of 1920, the epidemic did not assume such large proportions, at least, so far as the experiences in Moscow would indicate. Pappenheim gives a brief summary of the reports from Moscow. Tarasewitsch reported 22 cases; ages 16 to 65 years; mortality 25 percent. Other writers reported individual cases, but no complete review of the epidemic in Moscow in 1920-21 was known to Pappenheim. Kononowa is quoted in the report of the pathological findings of a woman with encephalitis who died in the fourth month of pregnancy. The embryo showed characteristic changes in the gray matter of the third and fourth ventricles and the aqueduct of Sylvius.

An epidemic of encephalitis in Moscow, 1922-23, has been described by Margouliss (M 74). Forty-two cases were seen at the Clinical Institute (Table 1). Six were fatal (14.3 percent). The ages were from seven to forty years.

These patients were with others in the ward for nervous diseases, but there was no instance of contagion.

Table 1

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN MOSCOW [1922-23, MARGOULISS]

1922					1923							
Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	Male	Female
1	2	1	3	4	16	7	5	0	1	2	27	15

There were also cases in other clinics in Moscow, bringing the total to 123 for this epidemic.

Later, Margoulis and Model (M 79) gave their findings on fifty cases observed at the Babuchin Hospital, Moscow, during the years 1926-27 (Table 2).

Table 2

AGE AND SEX DISTRIBUTION OF 50 CASES IN MOSCOW [MARGOULIS AND MODEL]

Age Group					
Under 20 yrs.	20-30 yrs.	30-40 yrs.	40 yrs. and over	Male	Female
7	21	16	4	34	16

The epidemic of lethargic encephalitis reached Western Siberia in the autumn of 1919, according to Skalsky (S 234a), and reached its height there in 1922 to 1924.

Table 3

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN RUSSIA [1923-1928]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1923	....	....	....	....	....	....	....	....	78	92	62	61	960
1924	146	147	184	147	138	157	252	130	27	119	123	180	2,076
1925	169	142	251	212	263	214	217	164	195	190	128	125	2,132
1926	235	270	267	119	142	136	156	124	141	83	115	115	2,362
1927	136	107	173	110	127	109	106	100	86	89	82	70	1,296
1928*													

* To February 29, 58 cases in the Ukraine.

From the *League of Nations Monthly Epidemiological Reports.*

The *League of Nations Monthly Epidemiological Reports* give the figures shown in Table 3 for the incidence of epidemic encephalitis in Russia from September, 1923, to February, 1928.

Reports of only sixty cases have been collected from the literature. These have been analysed in Table 4.

Table 4

AGE, SEX AND SEASONAL INCIDENCE OF 60 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group						Quarter				
		Male	Female	1–9	10–19	20–29	30–39	40–49	60–69	I	II	III	IV	N.S.
1919	7	5	2	...	2	1	2	2	...	...	1	1	4	1
1920	11	4	7	1	2	7	1	...	...	4	1	2	2	2
1921	15	9	6	1	3	6	3	2	...	3	2	5	2	3
1922	5	3	2	...	1	3	1	...	...	1	1	...	2	1
1923	10	9	1	2	1	7	...	...	...	...	5	...	2	3
1924	4	4	.....	...	1	1	2	...	...	3	1			
1925	5	2	3	...	...	2	2	...	1	...	1	1	1	2
1926	2	2	.....	...	...	2	...	...	...	2				
N.S.	1	.....	1	...	...	1	...	...	...	...	...	...	...	1
Total	60	38	22	4	10	30	11	4	1	13	12	9	13	13

Greece

Dr. Caryophyllis of Athens observed two cases of epidemic encephalitis in October, 1918. A description of these cases has been given by Netter (N 47).

Triminakis (T 96) reports six chronic cases, in two of which, one in Athens and one in Mytilene, the acute onset occurred in 1918.

The epidemic in Greece, according to Cawadias (C 72), began in January, 1920. He offers no suggestions as to the method by which the disease may have been introduced into the country, and gives no account of the course of the epidemic.

Reports of only eleven cases occurring in Greece have been collected from the available literature (Table 1).

Table 1

Age, Sex and Seasonal Incidence of 11 Cases Reported in the Literature

Year	Total	Sex		Age group				Quarter				
		Male	Female	10–19	20–29	30–39	50–59	I	II	III	IV	N.S.
1918	4	3	1	2	2	.....	.....	...	...	...	2	2
1920	3	3	.....	1	.....	2	.....	3				
1921	1	1	.....	1	.....	.....	.....	...	...	...	...	1
1922	1	.....	1	.....	.....	1	.....	...	...	...	...	1
1923	2	1	1	1	.....	.....	1	1	...	...	1	
Total	11	8	3	5	2	3	1	4	...	...	3	4

The League of Nations gives the following figures for cases and deaths from 1923 to 1928 (Table 2).

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN GREECE [1923-1928]

Year		Cases	Deaths
1923	Jan.	40	
	Feb.	23	
	Mar.	39	
	Apr.	18	
	May	11	
	June	1	
1924		11	2
1925		10	
1926		1	
1927*		3	
1928*			

* To June 15.

From the *League of Nations Monthly Epidemiological Reports.*

SPAIN AND PORTUGAL

Spain.—The "Meningo-encephalitic epidemic" began in Spain in the winter of 1919-20, according to Maranon (M 65). There were one hundred cases in Valence and fifty in Madrid, with smaller numbers in other localities. Although some cases occurred in close proximity to each other, no definite case of contagion could be verified. The death rate, ten percent, was low.

Fernandaz-Sanz (F 27) reported twenty-five cases that had come under his observation from the middle of December, 1919 to the end of February, 1920, and made the comment

that the disease had spread over all parts of Spain. Ten of his patients were from Madrid, and one each from Avila, Toledo, Ciudad Real, Cáceres, Murcia, Granada and Almería. Of these seventeen cases, eleven were men. There were three deaths in the group. The contagiousness of the disease is discussed in a later article (F 28a). Two of the author's cases seem to show possible contagion. A mother, who had nursed her daughter during the first five days of the latter's illness, was taken sick fifteen days after the onset of the daughter's illness.

Portugal.—An account of the early years of the epidemic in Portugal has been given by Jorge (J 60). The first reported case was seen in Lisbon in February, 1919, by Assis Lopes and Cancella D'Abreu, and was described in *A Medicina contemporanea,* May, 1919.

By March, 1919, only seven cases had been reported in Portugal: these were from Lisbon and two were fatal. At the end of 1919, another was seen at Ponta Delgada on Ile S. Miguel. Early in 1920, Professor Tiago d'Almeda reported two cases at Porto. There was one case at Beja, and Geraldes Cardosa saw one at Castello Branco.

Epidemic encephalitis was made reportable in Portugal beginning June 1, 1920, but the peak of the epidemic of this year was in April and May. "All districts were affected; from north to south, the territory was invaded by the infection. Cases appeared suddenly without zones of confluence nor avenues of propagation. All at once invasion of the communes contiguous to Lisbon was reported. In every locality there was the same phenomenon of dispersion; cases appeared here and there, scattered through the population without visible connection or preference for localization."

However, the total number of cases reported was small.

At the most there were two hundred up to December, 1920. The actual figure was thought to be higher, counting benign cases unrecognized, and cases not reported.

In a series of 65 cases observed in Portugal, 20 were fatal (30.7 percent). Inclusion of benign or abortive cases would render this percentage lower.

There was no difference in the incidence in town or country. No social or professional preference was observed.

Fifty-eight cases reported from the literature of Spain and one from Portugal are grouped in the following table.

AGE, SEX AND SEASONAL INCIDENCE OF 59 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group								Quarter				
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	I	II	III	IV	N.S.
Spain																
1918	1	1	.....	....	.....	...	1	...	...	...	...	..	..	...	...	1
1919	1	1	.....	....	.....	...	...	1	...	...	...	...	...	...	1	
1920	32	20	12	....	.....	...	6	5	10	7	4	28	1	2	1	
1922	4	4	.....	....	1	1	...	...	2	...	...	1	1	...	...	2
1923	1	1	.....	....	.....	1	...	...	...	...	...	...	...	...	...	1
1924	3	3	.....	....	.....	...	3	...	...	...	...	..	..	...	1	2
1925	3	1	2	....	.....	1	1	...	1	...	...	2	..	1	...	
N.S.	13	4	6*	3	.....	5	1	4	2	1	...	..	..	...	...	13
Total	58	35	20	3	1	8	12	10	15	8	4	31	2	3	3	19
Portugal	1	1	.....	....	.....	...	...	...	...	...	1	..	1			

In a case observed by Novaes and Sousa, an infant was born with myoclonic convulsive movements similar to those of the mother, who was suffering from epidemic encephalitis at the time of delivery. The myoclonic movements of the infant decreased and disappeared in five days.

Another case of possible contagion is quoted from the description of Cancella d'Abreu. A man living in Amadora (a suburb of Lisbon) had visited one of his parents, suffering from encephalitis, five days previous to his own illness.

United States

The first case of epidemic encephalitis to be reported in the United States was observed by Josephine B. Neal in New York City in September, 1918 (N 21). In October, 1918 four other cases were reported in New York by Neal (N 21), one in Iowa by Ely (E 32), and one in Camp Lee, Petersburg, West Virginia, by Pothier (P 201). During November, additional cases were reported in New York, one in Chicago by Bassoe (B 59), one in Texas, and one in Louisiana by H. F. Smith (S 243).

H. F. Smith, in his study of the outbreak in the United States during 1918 and 1919, found that there was a gradual extension of the disease from east to west; the peak in New York City was reached during January, 1919, in Virginia during February, and in Louisiana, Illinois and Texas during March; whereas in California more cases were reported during April than in any other one month.

Unrecognized cases may, however, have occurred in the west at an earlier date. In 1921 and 1922, Richards (R 108) observed sixteen patients in San Francisco with psychotic

sequelae of epidemic encephalitis. The initial illness in six of these cases was thought to have occurred during the period between September and December, 1918.

Tables 1 and 2 are taken from the above-mentioned report by H. F. Smith.

Table 1

Occurrence in the United States

Number of Cases Reported from Each State, September, 1918, to May, 1919

Massachusetts	4	*Louisiana	26
Connecticut	1	*Texas	8
New Hampshire	1	*Arkansas	5
Rhode Island	1	Missouri	1
*New York	43	Iowa	1
*Pennsylvania	3	Oklahoma	3
*Virginia	25	*Illinois	88
Tennessee	4	*Ohio	22
North Carolina	5	California	9
*South Carolina	1	*Republic of Mexico	1
Georgia	2		
Alabama	1	Total report	255

The States with an asterisk (*) are those in which intensive studies were conducted.

From Smith: *Public Health Report* 36: Part 1, p. 215, 1921.

The chronological incidence of 153 cases investigated in the nine states in which intensive studies were carried out is given in Table 2.

The world-wide outburst of encephalitis in 1920 and the

Table 2

EPIDEMIC ENCEPHALITIS IN THE UNITED STATES DURING 1918 AND 1919, BY STATES AND BY MONTH OF ONSET

State	Number of cases by months									Total
	1918				1919					
	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	
New York	1	3	4	6	9	8	7	2	2	42
Illinois	.....	.....	1	1	9	18	26	7	3	65
Louisiana	.....	.....	1	.....	.....	2	17	1	.....	21
Texas	.....	.....	1	.....	1	2	3	1	.....	8
Virginia	.....	.....	.....	2	.....	4	3	.....	.....	9
Ohio	.....	.....	.....	1	.....	1	1	.....	.....	3
Pennsylvania	.....	.....	.....	.....	.....	.....	3	.....	.....	3
South Carolina	.....	.....	.....	.....	.....	.....	1	.....	.....	1
Republic of Mexico	.....	.....	.....	.....	.....	.....	.....	1	.....	1
Total	1	3	7	10	19	35	61	12	5	153

From Smith: *Public Health Reports* 36: Part I, p. 218, 1921.

years following was reflected in an increase of cases in the United States, the peak being reached in this country in 1923. The figures in Table 3 have been taken from the *United States Public Health Reports,* from the *League of Nations Monthly Epidemiological Reports,* and from the *Bureau of Census Mortality Statistics,* and while the reporting of cases was evidently incomplete and the data presented contain many inaccuracies, this table is all that can be offered to indicate the course of the epidemic in this country.

Tables 4, 5 and 6 give the incidence of epidemic encephalitis as reported in New York State, New York City and Chicago, respectively.

Table 3

Epidemic Encephalitis in the United States [1918-28]*

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Primary Cause	Contrib. Cause	Total
1918—Cases	....	....	....	....	....	....	....	....	1	3	7	10			21
1919—Cases	19	35	61	12	5	0	....	....	....	....	....	....			132
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	375	214	589
1920—Cases	174	220	205	155	102	69	73	49	47	28	29	48			1,199
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	1,070	435	1,505
1921—Cases	220	463	....	....	....	....	....	....	....	....	....	....			1,822
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	1,355	354	1,334
1922—Cases	....	....	....	....	....	....	....	....	....	....	....	....			1,333
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	1,268	16	1,242
1923—Cases	137	397	556	134	60	75	50	57	40	48	38	26			1,618
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	1,966	22	1,939
1924—Cases	62	48	42	71	41	51	54	47	44	51	54	110			675
Deaths	....	....	....	....	....	....	....	....	....	....	....	....	1,441	31	1,410
1925—Cases	97	82	61	59	53	48	59	39	67	44	39	51			699
Deaths†	....	....	....	....	....	....	....	....	....	....	....	....			1,630
1926—Cases	80	83	115	87	80	92	79	74	81	66	55	79			971
Deaths	114	119	148	146	149	112	110	104	81	71	73	86			1,499
1927—Cases‡	370														
1928—Cases‡	342														

* The apparent discrepancy between the numbers of cases and the numbers of deaths from this disease is due to the fact that deaths were reported with more completeness than cases.

† All deaths, 1,597. ‡ To April 30.

From the *United States Public Health Reports*, the *League of Nations Monthly Epidemiological Reports*, and the *Bureau of the Census Mortality Statistics*.

Table 4

EPIDEMIC ENCEPHALITIS, NEW YORK STATE: CASES AND DEATHS, BY YEARS AND MONTHS

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1921													
Cases....	22	62	30	22	13	8	6	8	14	10	7	7	209
Deaths..	10	12	14	8	9	6	5	4	3	4	2	2	79
1922													
Cases....	4	6	8	16	17	6	10	7	6	16	5	6	107
Deaths..	5	5	9	6	9	4	9	1	2	3	5	5	63
1923													
Cases....	8	34	35	19	19	8	13	9	10	16	7	5	183
Deaths..	3	8	14	13	9	6	9	9	4	10	1	5	91
1924													
Cases....	17	5	6	8	9	12	11	4	8	7	12	26	125
Deaths..	10	4	5	5	6	8	6	4	7	3	10	6	74
1925													
Cases....	26	18	16	6	16	12	9	6	12	6	8	5	140
Deaths..	11	10	4	8	10	8	7	5	1	5	7	9	85
1926													
Cases....	5	8	15	12	5	12	9	8	7	2	3	7	93
Deaths..	5	3	13	7	6	4	7	7	7	2	4	5	70
1927													
Cases....	6	6	4	2	5	3	8	7	5	6	5	5	62
Deaths..	5	3	3	2	5	1	9	4	7	5	4	3	51
1928													
Cases....	10												
Deaths..	8												

From *N. Y. State Monthly Vital Statistics Review.*

Table 5

Incidence and Mortality of Epidemic Encephalitis New York City [1919-28]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total	Percentage case fatality
1919														
Cases	5	16	25	10	16	4	2	1	8	23	9	9	118	
Deaths	....	....	9	1	5	1	1	...	5	7	3	1	34	29
1920														
Cases	36	149	116	66	42	20	39	28	22	11	12	24	573	
Deaths	11	75	57	30	28	13	11	5	6	4	8	12	260	45
1921														
Cases	61	195	77	29	40	22	29	11	27	9	12	16	528	
Deaths	25	24	41	19	21	16	15	6	13	7	6	6	194	37
1922														
Cases	14	29	85	196	93	49	20	36	24	27	11	27	611	
Deaths	15	11	24	45	20	19	12	19	14	8	3	11	204	33
1923														
Cases	56	274	247	97	54	40	27	41	13	21	28	25	923	
Deaths	15	73	77	34	34	21	14	21	18	14	20	6	349	38
1924														
Cases	22	24	51	60	57	29	18	32	27	37	58	50	465	
Deaths	16	17	16	29	26	21	15	12	26	16	24	28	247	53
1925														
Cases	49	42	30	23	22	22	13	29	23	33	20	21	327	
Deaths	25	17	20	19	13	14	10	18	12	13	10	11	185	57
1926														
Cases	30	26	46	30	25	34	24	25	17	11	11	18	297	
Deaths	13	21	19	24	23	11	15	20	4	13	9	11	183	62
1927														
Cases	20	23	24	20	30	14	16	11	16	23	19	16	232	
Deaths	19	10	16	18	12	14	16	10	16	11	7	12	161	69
1928*														
Cases	105							...	...	...	...	...	....	
Deaths	63							...	...	...	...	...	....	60

* First 6 months.

From the *City of New York, Department of Health, Weekly Bulletin,* the *New York State Monthly Vital Statistics Review* and Jordan, *Epidemic Encephalitis.*

Table 6

Epidemic Encephalitis, Chicago [1919-28]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1919													
Cases....	0	0	27	9	3	0	1	0	1	3	9	16	69
Deaths..	0	0	9	5	0	1	0	1	0	1	3	5	25
1920													
Cases....	17	26	13	12	10	3	2	1	1	0	0	5	90
Deaths..	10	10	8	8	2	2	2	3	1	0	2	1	49
1921													
Cases....	21	25	10	5	4	1	0	0	0	0	1	0	67
Deaths..	11	16	5	2	3	1	2	2	2	3	0	2	49
1922													
Cases....	4	12	6	8	0	5	2	5	4	5	3	4	58
Deaths..	1	3	2	2	3	0	0	0	0	3	0	2	16
1923													
Cases....	8	10	7	11	4	6	3	5	4	2	8	2	70
Deaths..	3	2	1	2	0	4	1	1	1	0	2	2	19
1924													
Cases....	4	0	5	4	2	4	4	0	4	1	7	13	48
Deaths..	1	0	0	0	1	0	3	0	1	0	2	2	10
1925													
Cases....	11	5	8	5	1	2	1	4	8	0	4	0	49
Deaths..	3	2	0	4	0	2	0	1	2	2	2	1	19
1926													
Cases....	2	1	1	4	3	2	4	4	4	5	2	2	34
Deaths..	0	1	1	2	0	2	0	1	2	4	1	2	16
1927													
Cases....	4	3	5	11	7	2	4	6	2	2	2	1	49
Deaths..	1	0	0	5	1	2	1	4	1	0	0	0	15
1928													
Cases....	0	4	3	2	2	8	1	1	1	0			
Deaths..	0	1	0	1	1	5	1	1	1	0			

From the Dept. of Health, City of Chicago, personal communication from Arnold H. Kegel, Commissioner of Health.

Certain groups of cases have been studied in more or less detail with regard to age and sex incidence and mortality. The Public Health Service conducted a survey (74) of the occurrence of epidemic encephalitis in the United States for the period from September, 1918 to May, 1919. The age distribution of 181 cases included in this study is given in the following table:

Table 7

AGE DISTRIBUTION OF 181 CASES OF EPIDEMIC ENCEPHALITIS IN THE UNITED STATES

Age group (years)	No. of cases	Percentage of total
Under 1	6	3.32
1–4	21	11.6
Total under 5	27	14.92
5–9	22	12.15
10–14	15	8.3
15–19	12	6.63
20–24	15	8.3
25–29	13	7.18
30–34	14	7.73
35–39	12	6.63
40–44	20	11.05
45–49	8	4.42
50–54	7	3.87
55–59	6	3.31
60–64	5	2.75
65–69	4	2.21
70–74		
75 and over	1	.55
Total 5 and over	181	100.00

From Smith: *Public Health Reports* 36: p. 319, 1921.

The case fatality rate was 29 percent.

The age distribution of 274 cases observed by Dr. Neal (N 25) in New York City from the beginning of the epidemic to May, 1921, is shown in Table 8.

Table 8

AGE DISTRIBUTION OF 274 CASES OF EPIDEMIC ENCEPHALITIS
[NEAL, NEW YORK CITY]

Age	No. of Cases
0–3 months	4
3–6 "	4
6–12 "	8
1–2 years	20
2–5 "	29
5–10 "	47
10–15 "	25
Sub-total	137
15–20 years	15
20–30 "	56
30–40 "	33
40–50 "	24
Over 50 "	9
Total	274

The mortality rate was 28 percent. Fifty percent of the cases in this series were under fifteen years, a much higher proportion than that usually noted. This is accounted for by the author by the fact that a large number of children were seen in consultation for diseases of the central nervous system.

Happ and Mason (H 63) reported a series of eighty-one cases in Baltimore observed during the period from February, 1919, to February, 1921. A classification of their cases gives the following:

Table 9

AGE AND SEX DISTRIBUTION OF 81 CASES [HAPP AND MASON, BALTIMORE]

Age	Male	Female	Total
0–3 months	1	2	3
3–6 "		1	1
6–12 "			
1–2 years	1	1	2
2–5 "	5	4	9
5–10 "	10	8	18
10–15 "	4	2	6
Sub-total	21	18	39
15–20 "	2	4	6
20–30 "	6	5	11
30–40 "	9	2	11
40–50 "	6	2	8
50–60 "			
60–70 "	1		1
Total	45	31	76

Deaths, 6 (7.4 percent). Here, again, about 50 percent of the cases were under fifteen years.

Epidemic encephalitis cases and deaths in New York City by age and sex, 1920 (*City of New York, Department of Health Weekly Bulletin,* n. s. 10: no. 12, page 89, Mar. 19, 1921), are tabulated in Table 10.

Table 10

EPIDEMIC ENCEPHALITIS, CASES AND DEATHS BY AGE AND SEX, 1920

Age	Cases			Deaths		
	Male	Female	Total	Male	Female	Total
Under 5 yrs.	23	24	47	10	8	18
5–9 yrs.	26	14	45	10	2	12
10–14 "	32	18	50	6	5	11
15–19 "	23	24	47	5	5	10
20–24 "	37	34	71	13	11	24
25–29 "	29	27	56	11	14	25
30–34 "	35	18	53	8	8	16
35–39 "	30	18	48	14	11	25
40–44 "	19	14	33	7	10	17
45–49 "	24	16	40	13	7	20
50–54 "	15	8	23	8	3	11
55–59 "	8	3	11	6	1	7
60–64 "	7	7	14	3	5	8
65–69 "	4	4	8	2	1	3
70–74 "	3	2	5	2	1	3
75–79 "	0	1	1	0	1	1
80–84 "	1	1	2	0	0	0
Totals	316	233	549	118	93	211

From the *City of New York, Department of Health, Weekly Bulletin.*

Strauss and Wechsler (S 384) made a study of 864 cases (Table 11). About 200 cases were observed by the authors and the records of the others were obtained from thirty neurologists in the United States and Canada. More than half of the cases occurred in New York City.

Table 11

AGE INCIDENCE OF 864 CASES [STRAUSS AND WECHSLER, UNITED STATES AND CANADA]*

Age group	Cases	Percentage
Under 5 yrs.	13	1.5
6–10 yrs.	35	4.
11–20 "	136	15.7
21–30 "	222	25.7
31–40 "	215	25.
41–50 "	140	16.2
51–60 "	72	8.3
61–70 "	23	2.8
Over 70 yrs.	8	0.7
Total	864	100.0

* Age incidence, 4 weeks to 84 years.

The total number of deaths was 177, or 20.5 percent.

The incidence by sex in this series was as follows:

Male, number of cases, 522, percentage 60.5; female, number of cases, 342, percentage 39.5.

Every conceivable occupation was represented; it was noted that the number of physicians was seventeen, or 2 percent.

Grosman (G 154) has described the sequelae of epidemic encephalitis as based on his study of 145 cases in New York City. The mortality rate in this series was 20 percent.

From Spokane, Washington, Price (P 215) reported 78 cases seen from June, 1919 to March, 1921.

There were 19 deaths (24 percent), 13 of which were in patients over 40 years. Only one death occurred in a patient under 20 years.

Table 11a

AGE INCIDENCE OF 78 CASES
[PRICE, SPOKANE, WASHINGTON]*

Age group	Cases
First decade	5
Second "	11
Third "	14
Fourth "	10
Fifth "	18
Sixth "	12
Seventh "	5
Eighth "	2
Ninth "	1

* Age incidence, 16 months to 82 years.

Another group of cases in the northwest has been described by House (H 239). This author observed 145 patients in Portland, Oregon; 90 were examined during the acute stage and 55 during the chronic stage or because of sequelae. The mortality rate was between 26 and 44 percent. The epidemic in this locality occurred between October, 1919 and January, 1922, and moved in waves. The first wave came during October, November and December, 1919, with lesser waves in January and March and with complete subsidence in May, 1920. Fifty-four acute cases were examined during this period. From October, 1920 to April, 1921, there was an almost continuous series of acute cases, 36 in all. These were less severe than those of the previous winter and the mortality rate was about one-half as high.

The *City of New York, Department of Health, Weekly Bulletin,* vol. 17, no. 28, 1928, gives the age grouping

of 531 cases in New York City during 1926 and 1927. Slightly over half the cases, or 55.45 percent, occurred in males (Table 12).

Table 12

AGE DISTRIBUTION OF 531 CASES IN NEW YORK CITY [1926-27]

Under 1 year		Under 5 years		All ages	
Age group	Cases	Age group	Cases	Age group	Cases
Under 1 mo. . .	0	Under 1 yr. . .	13	Under 5 yrs. . .	76
1–2	0	1–2.	18	5–9	38
2–3	1	2–3.	17	10–14.	38
3–4	1	3–4.	13	15–24.	88
4–5	1	4–5.	15	25–34.	82
5–6	2	. . . . .	. . . . .	35–44.	83
6–7	2	. . . . .	. . . . .	45–54.	70
7–8	2	. . . . .	. . . . .	55–64.	43
8–9	1	. . . . .	. . . . .	Over 65.	13
9–10.	3				
10–11.	0				
11–12.	0				
Total.	13	. . . . .	76	. . . . .	531

From the *City of New York, Department of Health, Weekly Bulletin,* vol. 17, no. 28, 1928.

The same *Bulletin* gives the total of cases reported to the Department of Health during the nine years 1919-27, as 4,074, and the total number of deaths as 1,817, a case fatality of 45 percent. The comment is made, however, that "The registered case fatality rate is probably too high for the notifica-

tion of cases is always incomplete, whereas the reports of deaths are practically complete."

One thousand, four hundred and twenty-three cases reported in the literature as occurring in the United States from 1918 to 1926 have been classified according to age, sex and seasonal incidence in Tables 13a and 13b.

Table 13a

AGE AND SEX INCIDENCE OF 1,423 CASES REPORTED IN THE LITERATURE [1918-26]

Year	Total	Sex			Age group									
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.
1918	44	26	18	....	.....	5	11	9	7	7	3	1	...	1
1919	372	246	125	1	5	50	75	76	65	54	29	10	...	8
1920	447	276	167	4	3	56	103	112	78	50	27	3	1	14
1921	103	61	42	....	1	14	24	25	25	10	3	...	...	1
1922	81	50	30	1	.....	14	24	9	20	6	7	...	...	1
1923	82	46	33	3	1	14	32	8	10	8	2	3	1	3
1924	40	32	7	1	.....	4	14	8	6	3	5			
1925	13	7	6	....	.....	1	3	3	2	1	...	1	1	1
1926	7	3	4	....	.....	2	...	1	1	2	1			
N.S.	234	145	85	4	5	25	48	49	55	22	18	7	2	3
Total	1,423	892	517	14	15	185	334	300	269	163	95	25	5	32

Another number of the *Weekly Bulletin* (volume 17, no. 24, June 16, 1928, page 46) compares the "Death Rates per 100,000 Population, 19 Large American Cities, by Important Causes of Death, 1927." The figures for encephalitis lethargica are given in Table 14.

Table 13b

SEASONAL INCIDENCE OF 1,423 CASES REPORTED IN THE LITERATURE [1918-26]

Year	I	II	III	IV	N.S.
1918	9	3	2	27	3
1919	168	35	29	118	22
1920	284	82	27	41	13
1921	57	17	10	10	9
1922	37	17	7	14	6
1923	38	12	17	7	8
1924	7	13	4	13	3
1925	4	4	4	.....	1
1926	2	3	.....	2	
N.S.	.....	.....	.....	.....	234
Total	606	186	100	232	299

Table 14

DEATH RATE PER 100,000 POPULATION OF 19 AMERICAN CITIES IN 1927

Chicago	.39	Buffalo	.73
Philadelphia	.83	Milwaukee	.55
Detroit	1.20	Washington	1.48
Los Angeles	.67	Newark	1.50
Cleveland	1.02	Minneapolis	3.17
Baltimore	3.05	New Orleans	1.15
Boston	1.51	Cincinnati	1.22
Pittsburgh	1.05	St. Paul	2.01
San Francisco	2.26	Providence	.74
New York City	2.70		

Canada

The literature contains but few references relating to the incidence of epidemic encephalitis in Canada. Two outbreaks in Winnipeg have been described by Boyd (B 348, B 349, B 351.) The first occurred in the winter of 1919-20, with 159 cases in Manitoba, 89 of which were in Winnipeg; the mortality rate was 39 percent. The onset of the first case was during the last week of October, 1919. The ages were from 18 months to 72 years.

The second Winnipeg outbreak occurred in the winter of 1922-23, with 165 cases in all, 108 of which were in Winnipeg; the mortality rate was 25 percent.

Two hundred Canadian cases (including sixteen of Boyd's cases) reported in the literature have been classified with respect to age, sex and seasonal incidence (Tables 1a and 1b).

Table 1a

Age and Sex Incidence of 200 Cases Reported in the Literature [Canada]

Year	Total	Sex			Age group							
		Male	Female	N.S.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	N.S.
1919	17	9	8	....	2	.....	4	2	3	1	2	3
1920	6	6	.....	.....	...	2	1	.....	1	.....	1	1
1921	11	4	7	....	...	3	3	.....	2	.....	1	2
1922	1	.....	1	....	...	.....	1					
1923	146	1	.....	145	...	.....	1	.....	.....	.....	.....	145
N.S.	19	14	5	....	...	1	4	3	3	1	.....	7
Total	200	34	21	145	2	6	14	5	9	2	4	158

Table 1b

SEASONAL INCIDENCE OF 200 CASES REPORTED IN THE LITERATURE [CANADA]

Year	Quarter				
	I	II	III	IV	N.S.
1919	2	1		14	
1920	5	1			
1921	4		3	4	
1922	1				
1923	146				
N.S.					19
Total	158	2	3	18	19

The Department of the Public Health of Nova Scotia reports the incidence in that province as follows:

Table 2

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN NOVA SCOTIA

Years	1919	1920	1921	1922	1923	1924	1925	1926	1927
Cases reported	3	3	5	1	4	2	5	3	1
Deaths, Male			2	1	2	1	1	1	
Deaths, Female			2	1	1	1	0	1	

From the Department of Public Health, Nova Scotia.

The record of deaths by years and months as received from the Dominion Bureau of Statistics is given in Table 3.

Table 3

MORTALITY FROM EPIDEMIC ENCEPHALITIS [1921-27, CANADA]

	1921	1922	1923	1924	1925	1926	1927
Jan.	12	4	12	6	9	6	14
Feb.	13	8	30	8	17	6	9
Mar.	16	4	29	5	10	6	5
Apr.	11	5	15	9	7	7	8
May	8	3	14	10	14	11	17
June	6	5	7	11	7	9	11
July	8	3	8	12	11	7	10
Aug.	2	3	3	1	8	5	10
Sept.	7	2	9	4	11	5	11
Oct.	8	5	8	3	10	14	10
Nov.	5	2	5	5	6	13	7
Dec.	6	4	6	8	8	10	8
Total	102	48	146	82	118	99	120

From the Dominion Bureau of Statistics.

In 1919, according to the U. S. *Public Health Reports*, there were 78 cases and 20 deaths; and in 1920 there were 32 cases.

SOUTH AMERICA

Although it is generally stated that epidemic encephalitis was not reported from South America until 1919, Netter (N 47) refers to a note from Professor Morquio, who saw three infants with this disease at Montevideo and reported the case in the *Rivista médical de Uruguay*, in August, 1918.

In May, 1919, a case was observed in Argentina by Desté-

fano (D 107). During 1919, another case was seen in Buenos Aires, one in Brazil and four in Peru.

Ricaldoni (R 106) states that while isolated cases had appeared two or three years earlier, the disease became "epidemic" in 1920 and reached its maximum during the winter months, July and August. In Uruguay (in 1920?) there were 208 cases, with 98 deaths (over 47 percent), although the author believes that many light cases were not reported.

The figures in Tables 1 and 2 are taken from Ricaldoni's report.

Table 1

Mortality from Epidemic Encephalitis
[Ricaldoni]

Year		1920	1921	1922
Uruguay	Cases*	42	9	19
	Deaths	11	3	2
	Mortality per cent	26.2	33.3	10.5
Argentina Dr. Ballagtia	Deaths	47	25	
Rio de Janeiro	Cases	15	10	7
	Deaths	15	7	5

* Hospital admissions.

The seasonal incidence of the cases reported in Rio de Janeiro is given by quarters in Table 2.

Table 2

SEASONAL INCIDENCE IN RIO DE JANEIRO
[RICALDONI]

Year	Quarter			
	I	II	III	IV
1920	.....	.....	14	
1921	5	3	2	
1922	3	1	2	1

Espejo (E 52) has reported the incidence in Lima, Peru (Table 3); the age and sex are not given. The mortality rate was 13 percent.

Table 3

INCIDENCE IN LIMA, PERU, 1919-24
[ESPEJO]

Year	Quarter					
	I	II	III	IV	N.S.	Total
1919	1	1	1	1	.....	4
1920	3	6	3	2	.....	14
1921	.....	3	6	.....	1	10
1922	1	.....	3	3	.....	7
1923	3	2	4	.....	.....	9
1924	.....	3	.....	.....	.....	3
Total	8	15	17	6	1	47

The incidence in Uruguay, from 1922 to 1927, is given as follows in the *League of Nations Monthly Epidemiological Reports* (Table 4):

Table 4

INCIDENCE IN URUGUAY [1922-27]

Year	Cases	Deaths
1922	10	8
1923	3	3
1924	9	14
1925	4	1
1926	1	
1927	2	

From the *League of Nations Monthly Epidemiological Reports.*

An analysis of 104 additional cases, collected from the literature, is given in Tables 5a and 5b.

Table 5a

AGE AND SEX INCIDENCE OF 104 CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age Groups								
		Male	Female	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	N.S.
Argentina												
1919	2	1	1	.....	...	....	1	1				
1920	37	24	13	1	7	3	12	10	4			
1921	7	7	.....	.....	...	1	1	2	2	1		
1922	1	.....	1	.....	1							
1923	2	1	1	.....	...	1	1					
1924	6	3	3	.....	...	3	2	....	1			
1925	1	1	.....	.....	1							
1926	2	2	.....	.....	...	....	1	1				
N.S.	7	5	2	.....	3	2	1	1				
Total	65	44	21	1	12	10	19	15	7	1		
Peru												
1919	1	1	.....	.....	...	....	....	....	....	....	....	1
1920	10	7	3	.....	1	3	1	1	1	1	1	1
1921	6	5	1	.....	...	1	1	....	1	....	....	3
1922	3	3	.....	.....	...	1	....	1	1			
1923	5	5	.....	.....	...	....	1	3	1			
1924	2	.....	2	.....	...	....	....	....	....	2		
N.S.	2	2	.....	.....	...	....	....	1	....	....	1	
Total	29	23	6	.....	1	5	3	6	4	3	2	5
Brazil	8	3	5	.....	...	1	5	1	1			
Paraguay	1	1	.....	.....	1							
Uruguay	1	1	.....	.....	1							
South American Total	104	72	32	1	16	16	26	22	12	4	2	5

Table 5b

Seasonal Incidence of 104 Cases Reported in the Literature

Year	Quarter				
	I	II	III	IV	N.S.
Argentina					
1919	.....	1	.....	.....	1
1920	.....	16	17	4	
1921	.....	4	3		
1922	1	.....	.....		
1923	.....	1	.....	1	
1924	1	2	.....	3	
1925	.....	1			
1926	.....	.....	2		
N.S.	.....	.....	.....	.....	7
Total	2	25	22	8	8
Peru					
1919	.....	1			
1920	1	5	3	1	
1921	.....	4	1	1	
1922	.....	.....	1	1	1
1923	2	2	1		
1924	.....	2			
Total	3	14	6	3	1
Brazil	.....	2	4	2	
Paraguay	.....	.....	.....	.....	1
Uruguay	.....	.....	.....	1	
South American Total	5	41	33	14	10

India

A small group of cases of epidemic encephalitis appeared in Karachi City, India, in November and December, 1919. Seventeen of these cases were reported by Malone and Maitra (M 53). Stephan and Bulchandani (S 316) believe that this was the first appearance of the disease in India. Six patients with encephalitis were admitted to the hospital within three weeks. A number of cases had developed in the city by December tenth. Five out of six were males. The majority were between fifteen and forty years of age.

With this exception no outbreaks of encephalitis have been reported from India. Thirty-five cases described in the literature are analysed in the accompanying table.

China

Cadbury's case (C 3) is thought to have been the first instance of epidemic encephalitis reported in China. The patient was a Chinese boy of seven years taken sick in Canton in February, 1919. Lennox (L 127) described a case occurring in Peking in December of the same year. In March and April, 1920, Judson (J 68) saw two cases in Hwaiyuan. Wassell (W 22) gives a detailed history of another in Muchang in December, 1921. During the same month an officer was taken sick with epidemic encephalitis in a military camp in Changsha. Shibley (S 169) in reporting this illness mentions the fact that a similar case had occurred in the same camp but does not give the time relationship; nor does he make any statement with regard to direct contact.

From February, 1925 to December, 1926, Chien (C 112) collected thirteen typical cases of epidemic encephalitis at the Hunan Yale Hospital. There had been no cases here (at

Age, Sex and Seasonal Incidence of 35 Cases from India Reported in the Literature

Year	Total	Sex		Age groups								Quarter				
		Male	Female	1–9	10–19	20–29	30–39	40–49	50–59	60–69	N.S.	I	II	III	IV	N.S.
1919	26	20	6	.....	6	11	5	1	2	1	.....	1	.....	.....	25	
1920	1	1	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	.....	.....	1
1921	2	2	.....	.....	1	.....	.....	.....	.....	.....	1	1	.....	.....	1	
1922	1	1	.....	.....	.....	1	.....	.....	.....	.....	.....	1				
1924	3	.....	3	1	.....	1	1	.....	.....	.....	.....	.....	3			
1926	2	.....	2	1	1	.....	.....	.....	.....	.....	.....	.....	1	1		
Total	35	24	11	2	8	14	6	1	2	1	1	3	4	1	26	1

Changsha) in the interval since Shibley's case in 1921. These patients were all natives of the Hunan province. An analysis of these cases reveals the following. Sex: males 9, females 4; age: 4-36 years (5 between 4 and 20, 6 between 21 and 30, and 1 over 30 years); occupation: students 6, housewives 2, farmers 2, merchants 2, and no occupation 1.

One hundred cases of encephalitis were studied at the Peking Union Medical College and reported by Maximilian Pfister in 1926 (P 130) (Table 1). This author believes that accounts of the occurrence of epidemic encephalitis in China must be received with caution, as there are many opportunities for error in diagnosis. He does not consider the description of Cadbury's case sufficiently complete to make the diagnosis certain; the same is true, he think, of Judson's cases in Hwaiyuan. Lennox's case in Peking, he believes to be encephalitis.

Table 1

INCIDENCE AT THE PEKING UNION MEDICAL COLLEGE [PFISTER]

Year	1919	1920	1921	1922	1923	1924	1925 (to June)	Total
Cases...	5	12	8	20	23	26	6	100

In 1919, the first case occurred in March in Shanghai; there was one in July in Fuchow on the South coast; three in the fall in Peking, Paoting, and Taiyuan in Shansi.

The greatest incidence was during the winter months, from December to March, the period of dry cold with dust-winds; the next greatest was in August, the beginning of the dry season. In this connection, the author notes that the great Jap-

anese epidemic in 1924 occurred during an unusually dry summer and declined with the beginning of the rainy period.

An analysis of these cases gives the following data. Age: 5 months to 61 years, with more than half in the period 14-27 years; sex: male 80, female 20; nationality: Chinese 82, American 10, Russian 4, English 1, Japanese 1, Korean 1, French 1; occupation: students 27, housewives 10, merchants 9, missionaries 7, teachers 5, nurses 4, farmers 4, apprentices 3, servants 3, rikischakuli 3, officials 2, police 2, manual laborers 2, others 6, and no occupation 13.

Although the author does not discuss the question of contagion, two of the patients in his series were nurses who took sick in March, 1924. They had been employed on the same ward to which a physician's wife had been admitted with encephalitis in February.

Pfister discusses the routes of entry into China. There are three possibilities: 1) from America, across the Pacific and Japan; 2) the land route over Siberia; and 3) from the West, across India:

1) In America, the first cases were in the East, at the end of 1918. In the West, the disease appeared a year later. The first cases in Shanghai were in March, 1919.

2) In Russia, encephalitis appeared some time before 1919 but apparently reached Siberia only toward the end of that year. Trans-Siberian travel was at that time interrupted in many places and it seems probable that the first cases in Peking (in the fall of 1919) were brought by railroad from the south of China, especially as the first cases in Mukden and Charbin were reported somewhat later.

3) In favor of this route *i. e.,* from Europe across the Red Sea and over India to the south ports of China, is (a) the

fact that the first cases appeared in the port cities connected with the West, and (b) the fact that at the same time (July, 1919) cases appeared in Andaman, an English island-group half way between India and China. The same route can be traced for cholera. The original carriers were probably English soldiers, among whom the first cases were seen in Karachi, on the northwest coast of India. In Japan, encephalitis appeared at the same time as in China.

Watson (W 25) believes that there may be endemic areas for encephalitis in China. This writer was transferred in 1926 to a hospital in Yunnanfer, a town in the more or less isolated province of Yunnan. Here he found a number of out-patients who showed a typical post-encephalitic Parkinsonsim. Because the province was so situated as to make outside contact rare, it seemed possible that this was an endemic area for encephalitis. The cases came from different villages, some of them from one to three days' journey from the hospital. There was no reliable way of ascertaining the facts, but apparently there had been no epidemics of any size.

According to the patients' testimony, the duration of symptoms had been from six to twelve years. This would place the acute illness of one patient in 1915.

If this were a new disease, why did it not become epidemic and spread in China as it did in Europe and America? There is no evidence of such an epidemic in Yunnan. If it was brought into Yunnanfer by way of Indo-China, it is strange that there were no cases among the Cantonese in this province, as they travel much more freely than do the Yunnanese.

Twenty-one cases have been collected from the literature and analysed in Table 2.

Table 2

AGE, SEX AND SEASONAL INCIDENCE OF 21 CASES REPORTED IN THE LITERATURE

Year	Sex			Age group						Quarter				
	Total	Male	Female	1–9	10–19	20–29	30–39	40–49	N.S.	I	II	III	IV	N.S
1919	3	3	.....	1	...	1	1	...	....	2	....	....	1	
1920	2	2	.....	...	1	1	...	...	....	1	1	....	....	
1921	3	3	.....	...	2	1	...	...	....	1	....	....	2	
1924	5	2	3	...	1	1	1	...	2	3	1	....	1	
N.S.	8	8	.....	...	3	1	2	2	....	....	....	....	....	8
Total	21	18	3	1	7	5	4	2	2	7	2	....	4	8

JAPAN

The most complete available report on the incidence of encephalitis in Japan is that given by Kaneko and Aoki (K 14a). These authors, as a result of their own observations and of a study of the Japanese literature, distinguish between two types of encephalitis. The one, Type A, occurs in the winter months and presents a clinical picture similar to that of the European disease; the other Type B, is observed in late summer and early fall, and differs in clinical and epidemiological characteristics from the usual form observed in Europe and the United States. The division made by Kaneko and Aoki is fol lowed here and the figures given are taken from their review

Encephalitis Epidemica A

Only 223 cases of this type could be collected from the Japanese literature.

Seasonal Incidence.—Wada reported 41 cases occurring from December, 1919 to March, 1920; Naito, 32 in January, 1920 (?); and Kusuda 4 from December, 1919 (?) to April, 1920 (?). The distribution of 94 additional cases, in which the month of onset could be determined, is given in Table 1.

Table 1

INCIDENCE OF 94 CASES OF ENCEPHALITIS A BY MONTHS

Month	Cases	Month	Cases	Month	Cases	Month	Cases
Jan.	31	Apr.		July	1	Oct.	3
Feb.	24	May	3	Aug.	2	Nov.	8
Mar.	5	June		Sept.		Dec.	17
Total							94

Age and Sex.—Wada's 41 cases were "mostly of middle age;" males 27, females 14. Of 147 cases in the literature, the age and sex incidence is given in Table 2.

Table 2

AGE, AND SEX INCIDENCE OF 147 CASES OF ENCEPHALITIS A REPORTED IN THE LITERATURE

	Age period							Total
	To 10 yrs.	11–20	21–30	31–40	41–50	51–60	Over 60	
Male	6	23	30	23	10	5	2	99
Female	4	14	14	9	3	2	2	48
Total	10	37	44	32	13	7	4	147

Occupation.—Laborers and students predominated.

Mortality.—The mortality in Type A as reported by different authors is shown in Table 3.

Table 3

MORTALITY FROM ENCEPHALITIS A

Author	Cases	Mortality percentage
Wada	41	17.0
Ujihara	7	26.6
Kaneko	102	24.5
Kashwado	66*	34.8

* Collected from the literature.

Thus the seasonal incidence in the winter months, the age incidence showing the 10-30-year age period to be the one most affected, and the moderate mortality rate all correspond to the usual statistics for epidemic encephalitis as observed in Europe and America.

Encephalitis Epidemica B

Incidence and Mortality.—According to Kaneko and Aoki, small epidemics of this type of the disease have occurred during the hot season at frequent intervals since 1871. The mortality rate has usually been high. Figures since 1901 are given in Table 4.

Official figures probably give an unduly high mortality rate, as the milder cases are not always reported. Of 125 cases (including 12 that were abortive) observed by four writers, there were 42 deaths (33.6 percent).

Table 4

MORTALITY FROM ENCEPHALITIS B [1901-27]

Year	Place	Cases	Deaths	Mortality percentage
1901	Kagawa	21	9	42.9
1901	(Wakagama)	.....	119	60.0
1912	Okayama	476	293	61.5
1919	Okayama	52	38	73.
1922	Okayama	95	73	76.8
1924	Japan	6,949	4,169	59.99
1926	Fukuoka	20	8	40.
1927	Fukuoka	19	10	53.

The B type occurs in summer and early fall. The hotter the summer, the more likely this disease is to appear in August. It is especially widespread if drought accompanies the heat. Such conditions prevailed in certain of the affected provinces in 1924, at the time of the great epidemic. In other provinces in north Japan the spread of the disease was delayed.

Iimura and Katsumata give figures for 1924 from three provinces by five-day periods (Table 4a).

Table 4a

INCIDENCE OF ENCEPHALITIS B IN 1924* [IIMURA AND KATSUMATA]

Period	Aug. 1–5	Aug. 6–10	Aug. 11–15	Aug. 16–20	Aug. 21–25	Aug. 26–30	Aug. 31–Sept. 5	Total
Cases	72	301	454	403	238	78	33	1,579

* Three provinces.

Y. Aoki reports the incidence of 37 cases from smaller epidemics (Table 5).

Table 5

INCIDENCE OF ENCEPHALITIS B [AOKI]

Year	July	August			September			October	Total
		1–10	11–20	21–31	1–10	11–20	21–30		
1926...	....	1	.....	1	5	5	5	1	18
1927...	1	.....	11	4	2	.....	.....	1	19
	1	1	11	5	7	5	5	2	37

The mortality was greater in the higher age groups, as shown by Katsumata in Tokushima (1924) (Table 6).

Table 6

MORTALITY FROM ENCEPHALITIS B IN 1924 [KATSUMATA]

Age	Cases	Deaths	Still sick	Mortality percentage
Under 10 yrs.....	21	10	4	47.6
11–20 "	25	10	10	40.0
21–30 "	20	9	5	45.0
31–40 "	23	12	6	52.2
41–50 "	25	17	5	68.0
51–60 "	55	34	15	61.8
61–70 "	67	42	13	62.7
71–80 "	49	41	5	83.7
Over 80 "	14	13	1	92.9
Total.....	299	189	63	

The report of the Minister of the Interior gives the following: To September 5, 4,427 cases; to September 15, 5,873 cases; to September 29, 6,547 cases; and for the whole epidemic, 6,949 cases.

Age.—Elderly people are more frequently affected in this type of encephalitis than young people (Table 7). Kaneko and Aoki are inclined to believe that this condition is not so infrequent in young people as appears, but that it runs a milder course and thus is more often overlooked.

Table 7

Age Incidence in Encephalitis B in 1924

Age group as determined in 5 provinces in 1924			Yamada report 1924			Kaneko report 1924		
Age	Cases	Per-centage	Age	Cases	Per-centage	Age	Cases	Per-centage
Under 10	195	6.7	Under 10	53	19.7	Under 20	14	25.9
11–20	207	7.0	11–20	29	10.7	21–30	10	18.5
21–30	116	3.9	21–30	14	5.2	31–40	6	11.1
31–40	201	6.8	31–40	18	6.6	41–50	6	11.1
41–50	259	8.8	41–50	36	13.3	51–60	6	11.1
51–60	461	15.7	51–60	38	14.1	Over 60	12	22.2
61–70	668	22.7	Over 60	81	30.1			
71–80	646	22.1						
Over 80	183	6.2						
Total...	2,936	.....	.....	269	.....	.....	54	

Sex.—The sex incidence as reported during the epidemic of 1924 is given in Table 8.

Table 8

SEX INCIDENCE IN ENCEPHALITIS B IN 1924

Sex	Male	Female	Total	Ratio of Male to Female
Cases	1,941	1,538	3,479	1.26 : 1

Occupation and Social Status.—Iimura, Katsumata and Tadara classified the chief occupations represented in the epidemic of 1924:

Peasants	1,983
Business	242
Laborers	184
Fishermen	75
Officials	19
Nurses	12
Waiters	28
Students	14
Others	181
No occupation	557
Total	3,295

It was suggested that the incidence was highest among the peasants or farmers because of overwork and exposure to the hot sun while watering their rice fields during the drought. Students, on the other hand, were on vacations at this season. Moreover, they fall within the lower age groups where the incidence is lower.

Tadara distinguished four classes, A, B, C, and D, according to living conditions, and found Class C most affected (48.09 percent) and Class B next (39.66 percent).

Contagion.—Iimura (1924) reported 671 cases in 671 famalies in Toyama. Kuroda, among 49 cases in Kagawa,

found only one apparent familial infection, but seven cases of possible contagion. In Nagano there was no report of two cases in one family. Katsumata reported some cases of possible contagion. Kaneko described four cases of illness following contact with a patient suffering from encephalitis. In the epidemic of 1924, only a few physicians contracted the disease, and these were elderly and not in active practice.

The spread of this type of encephalitis is not eccentric, but it is extraordinarily rapid; cases appear thickly scattered over an entire district almost at the same moment. In certain districts, cases are observed almost every year, in summer and early fall, thus showing not only an epidemic, but an endemic or sporadic-endemic, character.

The *League of Nations Monthly Epidemiological Report* (R. E. 98, p. 9, January 15, 1927), gives the following statement with regard to the more recent appearances of epidemic encephalitis in Japan:

"An outbreak of encephalitis occurred in Japan between August and October, 1926. The geographical distribution was similar to that of the larger epidemic which occurred during the same months. The highest incidence was around the inland sea, particularly in Kagawa, Okayama, and Tottori. But while the case incidence in these three provinces in 1924 varied from 100 to 310 per 100,000, it was only from 13 to 15 per 100,000 during the recent outbreak. It appears that the provinces farther east and north have not been affected by the epidemic."

Statistics for the incidence of epidemic encephalitis in Japan, 1924-27, have been received from the Central Sanitary Bureau of Tokyo (Table 9).

Discussion.—The most important points of differentiation between Encephalitis Type A and Encephalitis Type B as given by Kaneko, are the differences in the seasonal and age

Table 9

INCIDENCE OF EPIDEMIC ENCEPHALITIS IN JAPAN, [1924-27]

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
1924												
Cases	....	....	....	....	2	7	87	3,957	2,493	210		
Deaths	....	....	....	....	1	3	56	2,196	1,473	203		
1925												
Cases	4	4	3	3	2	....	5	2	7	14	5	
Deaths	2	2	2	2	....	....	2	1	4	9	4	1
1926												
Cases	1	....	4	8	2	4	9	195	661	91	22	15
Deaths	2	....	4	7	3	4	8	107	337	140	31	26
1927												
Cases	6	....	4	1	1	1	1	232	659	35	10	3
Deaths	7	....	3	1	2	....	1	90	390	114	59	4

From the Central Sanitary Bureau, Tokyo.

incidence, in the mortality rate, and in the clinical course of the disease.

These authors admit, however, that cases of the A Type are sometimes seen in summer, and that, cases of the B Type not infrequently occur in the cold season, although no large epidemics of this form have ever developed in the winter.

The incidence of Encephalitis B was much greater in the higher than in the lower age groups during the 1924 epidemic, but Kaneko and Aoki, as stated above, believe that the condition is milder in young people and thus more often overlooked.

The Japanese epidemic was characterized by a high mortality (60 percent), but these authors suggest that an increase

in virulence may occur in other tropical and sub-tropical climates, and refer in this connection to the high mortality rate of the X-disease in Australia. It may also be mentioned here that other countries have also at times reported a high death rate for encephalitis. In England and Wales, for example, the case mortality was 75 percent in 1922, 58 percent in 1926, and 71 percent in 1927.

Meningeal manifestations are more marked in Encephalitis B; in this type, too, a delirious or hyperkinetic state is often observed, in contrast to the lethargy of Type A. The Parkinsonian syndrome is exceptional in the B form, whereas neurasthenic symptoms, such as headache, sleeplessness and irritability, frequently followed. A permanent disturbance of the psyche often resulted. This was especially noticeable in children. However, equally marked variations in the clinical manifestations of the disease are frequently observed in Europe and the United States, in groups of cases occurring in different years or in different localities.

Kaneko and Aoki are unable to deduce proof of difference from the pathological-anatomical findings in the two forms. Flexner (F 66) found that the pathological changes in specimens taken from the brains of fatal Japanese cases closely resembled those present in the European and American cases of epidemic encephalitis, and believed that there were "strong reasons for believing that the Japanese epidemic disease is of the nature of the European and American disease."

Although the Japanese authors quoted conclude that Encephalitis B is a special form of encephalitis closely related to Encephalitis A, it does not appear that there are satisfactory grounds for making a differentiation, either between these two types in Japan, or between the Japanese and the European or American form of the disease.

The fact that Kaneko and Aoki have been able to trace back over a period of years to 1901, or perhaps even to 1871, recurrent outbreaks of the condition which they have termed Encephalitis B, suggests the possibility that epidemic encephalitis may be endemic in Japan. It may be of interest to refer in this connection to Watson's suggestion with regard to endemic foci in China (q. v.).

Philippine Islands

In 1919, there was a mild outbreak of epidemic encephalitis in the Philippine Islands. Lantin and Vitug (L 59) reported eight cases seen at the Philippine General Hospital, three in September and five in October, 1919. Of the four fatal cases, the diagnosis was confirmed by autopsy in three instances, as reported by de Leon (L 130). The patients were all adult Filipinos, six being males and two females.

The first appearance of the disease in epidemic form was in the period from November, 1922 to the spring of 1923, with the peak in December and January.

Table 1

Incidence of Epidemic Encephalitis, 1922-23
[L. W. Smith]

	1922	1923			
	Dec.	Jan.	Feb.	Mar.	Apr.
Cases	40	21	14	9	3
Deaths	11	8	5	4	

L. W. Smith (S 245) describes 27 cases admitted to the Philippine General Hospital and also reports briefly on the epidemic in the Islands. Incomplete figures from the Health Service showed the incidence as given in Table 1.

The morbidity and mortality as shown by admissions to twelve hospitals is given in Table 2.

Table 2

MORBIDITY AND MORTALITY OF EPIDEMIC ENCEPHALITIS*

	Sex			Total	Mortality percentage
	Male	Female	N.S.		
Cases	43	12	15	70	41.4
Deaths	24	3	2	29	

* Figures from twelve hospitals.

The ages varied from 30 years to 49 years.

It was felt that this was a disease chiefly of adult males (18-30 years). The spread was mysterious. The first case seen in Manila was that of a sailor on one of the inter-island boats. This patient was immediately isolated. Cases appeared in all parts of the city; in only one instance were there two in the same family or the same house. Different classes of society were about proportionally affected. The mortality was high.

Only two towns outside of Luzon suffered, Zamboanga and Cebu. The latter had only two cases recognized clinically. These occurred at the same time as the epidemic in Manila, although Cebu is nearly 640 kilometers farther south, and is completely isolated except by boat. In Zamboanga, 960 kilometers south of Manila, the disease did not appear until Feb-

ruary. Sison (S 232) reported all the cases (39) seen at the Philippine General Hospital from November, 1922 to May, 1923. Eighty-seven percent of these were males. The mortality rate was 45 percent. The ages ranged from four months to 53 years; there were two cases under 10 years; 6 from 10-13 years; 19 from 15-25; 5 from 26-35; and 7 from 36-55.

This author believed that most of the cases were from the poorer classes. The majority came from Manila, perhaps because those from other parts of the islands were unable to get to the city. In Manila, the majority of the cases came from Tondo, the most crowded part of the city.

In Zamboanga, 18 cases occurred in 1923; in February, 5; in March, 9; and in April 4. At this season, it was very hot during the day and very cold at night. Aguilar and Rodriguez (A 66) made a clinical study of 13 of these. The age and sex incidence is given in Table 3.

Table 3

AGE AND SEX INCIDENCE IN 13 CASES [AGUILAR AND RODRIGUEZ]

Age Group	9–15	15–20	21–25	20–63	Sex	Male	Female	Total
Cases.....	5	2	4	2	Cases	8	5	13

"These were twelve Filipinos and one American. Of the thirteen cases there were six students, one teacher, one clerk, three laborers, one farmer, and one housewife. . . . ."

"The cases were widely distributed in different places in Zamboango . . . . none of the cases studied came from the same vicinity, except in two instances; but the residences of

Table 4

AGE, SEX AND SEASONAL INCIDENCE OF 40 CASES REPORTED IN THE LITERATURE

Year	Total	Sex			Age group							Quarter				
		Male	Female	N.S.	1–9	10–19	20–29	30–39	40–49	50–59	N.S.	I	II	III	IV	N.S.
1919	8	6	2	.....	.....	.....	.....	.....	.....	.....	8	.....	.....	3	5	
1920	5	5	.....	.....	.....	1	2	1	1	.....	.....	.....	1	2	.....	2
1922	1	1	.....	.....	.....	.....	.....	.....	.....	1	.....	.....	1			
1924	1	.....	1	.....	1	.....	.....	.....	.....	.....	.....	1				
N.S.	25	15	5	5	.....	.....	3	3	1	.....	18*	13	.....	.....	.....	12
Total	40	27	8	5	1	1	5	4	2	1	26	14	2	5	5	14

* 9–63 years.

these cases, in the barrio of Tetuan and in the City of Zamboanga, are far apart. There were also no two cases registered in the same house."

Forty additional cases reported in the literature are analysed in Table 4.

Australia

"The Mysterious Disease"

Australian cases of epidemic encephalitis, of the type known in Europe and the United States, were not reported until 1919. There appeared, however, in Australia in 1917 and 1918 an epidemic disease, the nature of which has been long debated. The most exhaustive account of this "Mysterious" or "X-disease," is that given by Cleland and Campbell (C 155). Through their courtesy an *Extract from the Report of the Director-General of Public Health, New South Wales, for the year ended* 31*st December,* 1917, has been made available for the use of this survey.

Although Cleland and Campbell have concluded that the condition then observed is not identical with epidemic encephalitis, their figures are included in this report, because of the interest the outbreak attracted, and because a comparison of the situation in Australia with that found elsewhere may prove of value in the consideration of the subject as a whole.

Cleland and Campbell called attention to the fact that the X-disease occurred in the dry, hot western portion of the state with marked preference for late summer and autumn months. The climatic conditions were similar to those described in Japan at the time of the great epidemic in 1924. The mortality rate in both countries was high. These points of similarity

have frequently been pointed out by Dr. Neal, (personal communication) and are discussed by Kaneko (K 14a) in his review of encephalitis in Japan.

Campbell's report on the clinical manifestations of this disease in Australia is based on the findings in 134 cases which occurred in New South Wales during the period from February to April, 1917, and from January to March, 1918. These cases were observed by doctors in the districts where the epidemic prevailed. Many of them are included in independent reports in the literature. A list of references is appended: Anderson (A 103); Breinl (B 370, B 371); Burnell (B 434, B 434a); Litchfield (L 280); and Mathewson and Latham (M 167).

The monthly incidence of 117 cases is shown in Table 1 (taken from Table 1 of the report mentioned above).

Table 1

Monthly Incidence of Cases [X-Disease, Campbell]

1917	Cases	1918	Cases	Total
Jan.	1	Jan.	6	7
Feb.	11	Feb.	30 (? 32)	43
Mar.	36	Mar.	13	49
Apr.	15	Apr.	2	17
Dec.	1			1
Total	64		53	117

Age and Sex Incidence.— Campbell's figures for age and sex incidence and mortality have been combined in Tables 2 and 2a.

Table 2

AGE INCIDENCE [X-DISEASE, CAMPBELL]

Age group	Under 1 yr.	1–5	5–10	10–15	15–20	20–25	25–30	30–35	35–40	40–45	45–50	Over 50	N.S.
Cases	6	54	23	9	3	1	2	4	4	4	5	9	10

Table 2a

SEX INCIDENCE AND MORTALITY [X-DISEASE, CAMPBELL]

Sex	Male	Female	N.S.	Total	Deaths	Mortality percentage
Cases	85	36	13	134	94	70

The oldest patient was sixty-eight. The age was not stated in 10 cases, 6 of whom were adults. "This analysis shows that infants and children under 5 years of age (nearly 50 percent of the total) were exceedingly prone to affection, and that after the age of 15 years susceptibility rapidly diminished." There were, however, 34 cases in persons over 25 years of age.

Mortality.—"Ninty-four patients died, 35 recovered completely, and 5 recovered, but with subsequent paralysis (3 cases), or signs of mental disorder (2 cases). In 85 of the fatal cases 59 were under 14 years of age, 12 under 2 years, and 4 under 1 year."

Period of Incubation.—"Our experimental observations in this disease show that in monkeys and other animals there is a definite period of incubation (usually 5 to 12 days); it is almost certain, therefore, that in the human subject there is

a corresponding period, but we have not sufficient data to fix its length."

Contagion.—Twenty-two instances of possible connection between cases, or of two or more cases in one family, are described.

Reports for 1919-27.—The European form of epidemic encephalitis has apparently attracted little attention in Australia. Figures for the few cases reported in the literature as well as

Table 3

AGE, SEX AND SEASONAL INCIDENCE OF EPIDEMIC ENCEPHALITIS IN AUSTRALIA, NEW ZEALAND AND THE SAMOAN ISLANDS, CASES REPORTED IN THE LITERATURE

Year	Total	Sex		Age group						Quarter			
		Male	Female	1–9	10–19	20–29	30–39	40–59	60–69	I	II	III	IV
Australia													
1919	2	2	.....	1	.....	1	.....	.....	.....	1	..	1	
1920	3	1	2	...	2	.....	.....	1	.....	2	1		
1921	1	1	.....	...	1	.....	.....	.....	.....	..	1		
1923	2	1	1	...	.....	2	.....	.....	.....	..	1	1	
1925	1	1	.....	...	1	.....	.....	.....	.....	..	..	1	
N.S.	1	.....	1	...	.....	.....	.....	1					
Total	10	6	4	1	4	3	.....	2	.....	3	3	3	
New Zealand*													
1919	11	8	3	...	1	.....	4	4	1	1	9	1	
Samoan Islands†													
1922	7	5	2	1	4	2	.....	.....	.....	..	7		

* Rossiter, C. B. (R 264) reports 10 of these cases with 3 deaths.

† Matheson, F. (M 166).

those for cases from New Zealand and the Samoan Islands are included in Table 3.

The figures for deaths reported in Australia for the years 1922 to 1927 are given in Table 4.

Table 4

EPIDEMIC ENCEPHALITIS, DEATHS REPORTED IN AUSTRALIA [1922-27]

Year	1922	1923	1924	1925	1926	1927
Deaths....	60	57	45	88	88	74

From the *League of Nations Monthly Epidemiological Report* (R. E. 116, July, 1928).

The figures for cases and deaths reported in New Zealand for the years 1920 to 1927 are given in Table 5.

Table 5

EPIDEMIC ENCEPHALITIS, CASES AND DEATHS REPORTED IN NEW ZEALAND [1920-27]

Year	1920	1921	1922	1923	1924	1925	1926	1927
Cases....	42	23	36	36	30	24	42	37
Deaths.....	.....	14	13	13	13	2	8	14

From the *League of Nations Monthly Epidemiological Report* (R. E. 116, July, 1928).

AFRICA

Although no pronounced epidemic of encephalitis has been reported from Africa, isolated cases and small groups have been noted from time to time, since 1918, both in the states

bordering on the Mediterranean and in the southern portion of the continent.

In April, 1918, Ardin-Delteil (A 128) described a case of encephalitis lethargica in an army nurse, at Algiers. Another case was reported from the same locality at the same time.

At a meeting of the British Medical Association in South Africa in March, 1919, Loeser (L 295) described five cases of "polio-encephalitis" (three of them fatal) which had occurred during February and March. In April the same writer reported (L 293) two similar cases in Europeans, a little girl and a boy of seventeen. The clinical features and the section in one autopsied case seemed to point undoubtedly to a diagnosis of a epidemic encephalitis.

Butt (B 469) saw 13 cases among the native laborers in a gold mine in the Transvaal during the period from April to July, 1920. There were 12 deaths (92.3 percent).

A review of the incidence of encephalitis lethargica in Morocco has been published by Pujol (P 224). According to this account, the first case, observed by Dr. Spéder, occurred in June, 1917. In November, 1918, Dr. Bouveret of Mogador saw a case in a European, recently returned from France. Another case in Bondenif was also seen in 1918. In January, 1919, a Franciscan father developed encephalitis soon after his arrival from France. This patient was reported by Pujol and Junquet from Bondenif. In all, thirty-three cases have been collected by Pujol. The largest group (21) was reported from Casablanca, where the first case occurred in 1921. Of the total number, five were "infants"; the ages of the others were between 12 and 60 years. A fatal issue is described as "rare," but the mortality figures are not given.

In 1920, six cases were reported from Alexandria, Egypt. Three were seen by Valassopaulo (V 3). Three were reported

Table 1

AGE, SEX AND SEASONAL INCIDENCE OF 20 CASES REPORTED IN THE LITERATURE [NORTH AFRICAN STATES]

Year	Total	Sex			Age group						Quarter				
		Male	Female	N.S.	10–19	20–29	30–39	40–49	50–59	N.S.	I	II	III	IV	N.S.
Algeria															
1918	1	1	.....	.....	...	1	...	...	...	.....	...	1			
1919	5	4	1	....	3	1	1	...	...	.....	...	.....	...	5	
1920	1	1	.....	.....	1	...	...	...	...	.....	1				
Total	7	6	1	....	4	2	1	...	...	.....	1	1	...	5	
Morocco															
1917	1	.....	1	....	1	...	...	...	...	.....	...	1			
1918	2	.....	.....	2	...	...	...	...	...	2	...	.....	...	1	1
1919	1	1	.....	....	...	...	...	1	.	....	1				
1920	3	1	2	....	...	2	...	...	1	....	2	...	...	1	
Total	7	2	3	2	1	2	...	1	1	2	3	1	...	2	1
Egypt															
1920	5	5	.....	....	1	3	...	...	...	1	5				
N.S.	1	1	.....	.....	...	1	...	...	...	.....	...	...	...	...	1
Total	6	6	.....	....	1	4	...	...	...	1	5	...	...	...	1
Grand Total	20	14	4	2	6	8	1	1	1	3	9	2	...	7	2

from the Military Hospital by Beretta (B 132) and by Gabri (G 3).

Twenty cases reported in the literature from the North African states and 21 from South Africa have been grouped in Tables 1 and 2.

Table 2

AGE, SEX AND SEASONAL INCIDENCE OF 21 CASES REPORTED IN THE LITERATURE [SOUTH AFRICAN STATES]

Year	Total	Sex		Age group				Quarter				
		Male	Female	1–9	10–19	40–49	N.S.	I	II	III	IV	N.S.
1918	1	1	.....	.....	.....	1	....	....	....	....	1	
1919	7	6	1	1	1	.....	5	5	2			
1920	13	13	.....	.....	.....	.....	13	....	8	4	....	1
Total	21	20	1	1	1	1	18	5	10	4	1	1

The figures given in Table 3 for the incidence of epidemic encephalitis on the continent of Africa have been taken from the *Public Health Reports* and the *League of Nations Monthly Epidemiological Report.*

Table 3

INCIDENCE OF EPIDEMIC ENCEPHALITIS ON THE CONTINENT OF AFRICA [1920-27]

State		1920	1921	1922	1923	1924	1925	1926	1927*
French Protectorate of Morocco	Cases	.....	.....	.....	.....	.....	.....	.....	5
Algeria	Cases	6	.....	12	7	4	0	1	0
Tunis	Cases	.....	.....	.....	.....	0	0	0	0
Cyrenaica	Cases	.....	.....	.....	.....	1	0	0	0
Southern Rhodesia	Cases	.....	.....	.....	.....	.....	4	1	
	Deaths	.....	.....	.....	.....	.....	2	0	

* Algeria, through April; Tunis, through March; Cyrenaica, through February.

From the *Public Health Reports* and the *League of Nations Monthly Epidemiological Reports*.

SUMMARY OF INCIDENCE

In reviewing the literature for this Survey a record was kept of all individual cases of epidemic encephalitis reported, under the following headings: place, date (of onset of illness), age, sex, occupation, mortality, and relation to other cases. A total of 7,876 cases was reported although the data were not complete in every instance. Table III (Chart IIII) showing incidence by months has been based on this total. Of this number, too, a fatal outcome was noted in 3,980 cases, or a mortality rate of 50.53 percent.

In addition to these individual cases, groups of cases were reported by many authors. When these groups had been analysed in the original article to show part or all of the data mentioned above, the author's figures were added in the appropriate column in our analysis, and the cases in which the desired information was not given were included in the column N. S. (not specified). In this way a total of 9,680 cases, reported from all countries from which literature has been available for this Survey, has been collected. These have been analysed according to age, sex and seasonal incidence in Table IX. A similar table has been compiled for each country separately, and will be found under the appropriate section.

In the absence of a uniform practice among the various countries in the reporting of cases of epidemic encephalitis, there is considerable uncertainty as to the true and complete record of incidence of this disease. In preparing Table X we have given as the year of greatest prevalence that one for each country for which we have found, either in official reports (O R) of cases or deaths, or in assembled reports of cases or deaths in medical literature (L R), with duplicates carefully eliminated, the largest number of cases (C) or deaths (D). Similarly the total incidence for the period stated in Table XI is given as it has been found, either of cases or deaths reported officially or in the literature.

Table IX

Age, Sex and Seasonal Incidence of 9,680 Cases Reported in the Literature*

Year	Total	Sex			Age Group										Quarter				
		Male	Female	N.S.	Under 1 yr.	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+	N.S.	I	II	III	IV	N.S.
1915	10	7	3	...	.....	1	....	3	4	1	...	...	....	1	2	4	2	2	
1916	48	37	8	3	.....	2	4	10	10	13	...	...	....	9	3	1	7	37	
1917	150	91	40	19	3	39	18	17	23	23	6	...	1	20	89	34	7	7	13
1918	669	321	266	82	5	83	102	115	81	58	43	18	2	162	171	127	25	268	78
1919	1,268	759	469	40	9	101	286	293	213	146	85	31	2	102	458	119	86	461	144
1920	4,550	2,330	1,676	544	14	347	899	969	688	465	197	70	18	883	3,258	632	194	262	204
1921	578	320	244	14	4	43	121	126	97	81	17	10	2	77	344	69	48	45	72
1922	354	199	143	12	2	27	76	71	64	30	18	2	....	64	139	63	33	78	41
1923	601	235	172	194	1	37	78	64	55	33	17	5	1	310	428	79	39	24	31
1924	614	373	231	10	3	26	43	59	30	20	16	9	....	408	80	364	86	58	26
1925	89	43	45	1	.....	11	20	22	10	8	10	2	2	4	29	18	13	15	14
1926	38	18	20	...	.....	6	7	9	7	5	3	1	....	.....	15	10	8	5	
1927	8	2	6	...	.....	....	2	4	1	1	...	...	.....	.....	2	.....	....	.....	6
N.S.	703	400	279	24	8	64	146	159	136	65	46	15	8	56	.....	.....	....	.....	703
Total	9,680	5,135	3,602	943	49	787	1,802	1,921	1,419	949	458	163	36	2,096	5,018	1,520	548	1,262	1,332
		8,737			7,584										8,348				
Percent		58.7	41.2	...	0.64	10.3	23.7	25.3	18.7	12.5	6.0	2.1	0.47	.....	60.11	18.21	6.56	15.11	

* Cases reported individually 7,876; deaths recorded 3,980; mortality percentage 50.53.

Table X

Year of Greatest Prevalence by Country

Country	Year of Greatest Prevalence	Cases (C), or Deaths (D)	Population[1]	Incidence (Cases or Deaths) per 100,000 Population
Algeria	1922 O R	12 C	5,800,974	0.21 C
Argentina	1920 L R	47 D	8,462,020	0.6 D
Australia	1925 O R	88 D	5,436,794	1.65 D
Austria	1920 L R	906 C	6,131,445	14.67 C
Belgium	1921 O R	248 C	7,684,272	3.33 C
Brazil (Rio de Janeiro)	1920 L R	15 C	1,250,000	1.20 C
Bulgaria	1924 O R	146 C	4,861,439	3.1 C
Canada[2]	1923 O R	146 D	8,772,000	1.87 C
Czechoslovakia	1923 O R	366 C	13,595,816	2.72 C
Denmark	1920 O R	226 C	3,289,195	6.83 C
England and Wales	1924 O R	5,067 C	37,885,242	13.19 C
Finland	1921 O R	1,095 C	3,331,814	33.3 C
France	1920 L R	840 C	39,209,766	2.14 C
Germany[3] (Prussia)	1924 L R	2,187 C	37,075,240	5.66 C
Greece	1923 O R	132 C	4,730,000	2.8 C
Holland	1925 O R	120 C	6,841,155	1.7 C
Hungary	1920 L R	19 C	7,840,832	0.025 C
Ireland[4]				
North Ireland	1924 O R	278 C	1,250,531 (1911)	22.4 C
Irish Free State	1925 O R	16 C	3,139,688	0.47 C
Italy	1920 O R	5,009 C	38,000,000 (1920)	12.5 C

Japan	1924 O R	6,949 C	77,005,510	8.70 C
New Zealand	1920 O R	42 C	1,218,913	3.44 C
Norway[5]	1921 O R	55 C	637,566	8.62 C
(cities only)			(cities only)	
Peru (Lima)	1920 L R	14 C	176,500	7.93 C
Poland	1920 L R	1,462 C	24,272,349	5.76 C
Portugal[6]	1920 L R	200 C	5,545,595	3.60 C
Rumania	1920 L R	61 C	17,393,149	0.34 C
Russia	1926 O R	2,362 C	131,546,045	1.82 C
Scotland[7]	1924 O R	631 C	2,363,878	25.39 C
(16 cities)			(16 cities)	
Spain[8]	1920 L R	32 C	20,783,844	0.14 C
Sweden	1921 O R	1,512 C	5,903,762	25.4 C
Switzerland	1920 O R	984 C	3,880,320	25.25 C
United States[9]	1923 O R	1,966 D	105,710,720	1.84 D
Uruguay	1920 L R	208 C	1,494,953	13.4 C

[1] The figures for population are taken from The Handy Reference Atlas of the World by John Bartholomew, Tenth Edition, 1923. The year on which the population is based stated in a few instances only, as indicated in the table.

[2] *Canada*. In the province of Manitoba 165 cases (L R) were reported for the winter of 1922–23. Population of Manitoba, 610,118.
Rate for this period in Manitoba 27.5.

[3] *Germany* (*Prussia*). Deicher reported 2,187 cases in Prussia in 1924 and 11,309 cases for the period 1919–24. Population of Prussia, 37,075,240.

[4] *Ireland*. Total population of Ireland (1911) 4,390,219.
Population of Northern Ireland (1911) 1,250,531; Cases reported in Northern Ireland in 1924, 278 } Rate 22.4.
Irish Free State, Population ("pending adjustment of boundaries"), 3,139,688.
Cases reported for 2 years only, 1925–26; 1925, 16 cases, rate 0.47.

[5] *Norway*. Official reports are for cities only. Population of cities 637,566.

[6] *Portugal*. 1920 Jorge reports in this way: the total number of cases was small, "at the most two hundred up to December, 1920."

[7] *Scotland*. Official report of 631 cases for 16 cities (*League of Nations Monthly Epidemiological Reports*). Population of 16 cities (largest) given in Atlas 2,363,878. Rate (based on population of 16 largest cities), 25.39.

[8] *Spain*. Maranon reported for the winter of 1919–20 100 cases in Valence and 50 cases in Madrid. On the bases of 150 cases the rate is 0.72.

[9] *United States*. Greatest number of cases reported in 1921, 1,822, except for 1921 and 1922 more deaths than cases were reported.
1921, cases 1,822, rate 1.70.
1923, deaths 1,966, rate 1.84.

Table XI

Total Known Cases or Deaths by Countries, 1915-28

Country	Period Covered by Reports	Total Known Cases or Deaths	Population[1]
Algeria	1920–1927 O R	30 C	5,800,974
Argentina	1920–1921 L R	72 D	8,462,030
Australia	1922–1927 O R	412 D	5,436,794
Austria	1915–1926 L R	1,393 C	6,131,445
Belgium	1919–1928 O R	522 C	7,684,272
Brazil (Rio de Janeiro)	1920–1922 L R	32 C	1,250,000
Bulgaria	1924–1927 O R	234 C	4,861,439
Canada	1921–1927 O R	715 D	8,772,000
Czechoslovakia	1922–1928 O R	876 C	13,595,816
Denmark	1919–1928 O R	1,035 C	3,289,195
England and Wales	1918–1927 O R	16,048 C	37,885,242
Finland	1920–1928 O R	1,559 C	3,331,814
France	1915–1927 L R	1,767 C	39,209,766
Germany[2] (Prussia)	1919–1924 L R	11,309 C	37,075,240
Greece	1923–1928 O R	157 C	4,730,000
Holland	1920–1928 O R	510 D	6,841,155
Hungary	1919–1924 L R	24 C	7,840,832
Ireland[3] North Ireland	1918–1927 O R	394 C	1,250,531 (1911)
Irish Free State	1925–1926 O R	19 C.	3,139,688
Italy	1920–1927 O R	8,078 C	38,000,000 (1920)

Japan	1924–1927 O R	8,963 C	77,005,510
New South Wales (X-disease)	Feb.-Apr. 1917 / Jan.-Mar. 1918 O R	134 C	2,103,836
Norway[4]	1919–1928 O R	155 C (Cities only)	637,566 (Cities only)
Peru (Lima)	1919–1924 L R	47 C	176,467
Poland	1917–1924 L R	2,208 C	24,272,349
Portugal	1919–1920 L R	207 C	5,545,595
Roumania	1915–1925 L R	89 C	17,393,149
Russia	Sept. 1923 / Feb. 1928 O R	8,826 C	131,546,045
Scotland[5]	1921–1927 O R	1,131 C (16 cities)	2,363,878 (16 cities)
Spain	1918–1925 L R	58 C	20,783,844
Sweden	1919–1928 O R	3,463 C	5,903,762
Switzerland	1920–1928 O R	1,643 C	3,880,320
United States	1918–1928 O R	11,148 D	105,710,720
Uruguay[6]	1920–1927 L R & O R	237 C	1,494,953

[1] The figures for population are taken from The Handy Reference Atlas of the World by John Bartholomew, Tenth Edition, 1923. The year on which the population is based is stated in a few instances only, as indicated in the table.

[2] *Germany (Prussia).* Deicher reported 2,187 cases in Prussia in 1924 and 11,309 cases for the period 1919–24. Population of Prussia, 37,075,240.

[3] *Ireland.*
- Total population of Ireland (1911) 4,390,219.
- Population of Northern Ireland (1911) 1,250,531
- Cases reported in Northern Ireland in 1924, 278 — Rate 22.4.
- Irish Free State, Population ("pending adjustment of boundaries"), 3,139,688.
- Cases reported for 2 years only, 1925–26, 1925, 16 cases, rate 0.47.

[4] *Norway* Official reports are for cities only. Population of cities 637,566.

[5] *Scotland.* Official report of 631 cases for 16 cities (*League of Nations Monthly Epidemiological Reports*). Population of 16 cities (largest) given in Atlas 2,363,878. Rate (based on population of 16 largest cities), 25.39.

[6] *Uruguay.* In this instance the number of cases reported in the literature for 1920 (208 cases) has been added to the number given in the official reports for 1922–27 (29 cases) to obtain the total (237 cases).

A 1 **Abadie and Hesnard**—Sur une forme psychique pure d'encéphalite épidémique. J. de méd. de Bordeaux, 91; 330, **1920.**

A 2 —, Encéphalite aiguë épidémique. Gaz. hebd. d. sc. méd. Bordeaux, 42: 43, **1921;** Bull. et mém. Soc. méd. et chir. de Bordeaux, 548, **1921.**

A 3 —, Syndrome parkinsonien séquelle d'encéphalite léthargique. J. de méd. de Bordeaux, 92; 29, **1921.**

A 4 **Abadie and Labuchelle**—Un cas de troubles mentaux consecutifs à une encéphalomyélite épidémique chez un enfant de dix ans. J. de méd. de Bordeaux, 94: 683, **1922.**

A 5 —, Encéphalite épidémique avec hémiplegie; hémiparesie spasmodique residuelle. J. de méd. de Bordeaux, 96: 683, **1924.**

A 6 **Abadie and Massé**—Syndrome parkinsonien post-encéphalique. Gaz. hebd. d. sc. méd. Bordeaux, 42: 501, **1921.**

A 7 **Abadie, Molin de Teyssieu and Labuchelle**—Un cas de troubles mentaux post-encéphalitiques. J. de méd. de Bordeaux, 94: 601, **1922.**

A 8 **Abadie, J. and Hesnard**—Sur un forme psychique pure d'encéphalite épidémique. Gaz. hebd. d. sc. méd. Bordeaux, 41: 314, **1920;** Bull. et mém. Soc. méd. et chir de Bordeaux, 317, **1920.**

A 9 **Abbruzzetti, A.**—Encefalite letargica ambulatoria? Riforma med. 36: 120, **1920.**

A 10 —, Encefalite letargica e sindrome di Benedikt. Riforma med. 36: 203, **1920.**

A 11 —, Sindrome lenticolare e parkinsonismo in encefalite letargica cronica. Med. prat., 6: 281, 321, **1921.**

A 12 **Abbruzzetti, A.**—Sopra un caso di encefalite letargica a decorsocronico. Policlin. (sez. prat.), 28: 767, **1921.**

A 13 —, Di un nuovo segno osservato nei parkinsoniani post-encefalitici. Med. prat., 7: 333, **1922.**

A 14 **Abella, E.**—Encephalitis letargica. Semana méd., 27: II, 283, **1920.**

A 15 **Abély, X.**—Posologie de l'uroformine dans l'encéphalite épidémique. Bull. Soc. clin. de méd. ment., 14: 57, **1926.**

A 16 **Abente Haido, F.**—[Blood picture and treatment of epidemic encephalitis.] Rev. méd. d. Uruguay, 24: 12, **1921;** abst. J. A. M. A., 76: 1051, **1921.**

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